THE TANGLED BANK AN INTRODUCTION TO EVOLUTION



CARL ZIMMER

Second Edition

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3 4 5 6 7 8 9 23 22 21 20 19 18

To Grace, for growing our garden together



IT IS INTERESTING TO CONTEMPLATE AN ENTANGLED

BANK, clothed with many plants of many kinds, with birds singing on the bushes, with various insects flitting about, and with worms crawling through the damp earth, and to reflect that these elaborately constructed forms, so different from each other, and dependent on each other in so complex a manner, have all been provided by laws acting around us. These laws, taken in the largest sense, being Growth with Reproduction; inheritance with is almost implied by reproduction; Variability from the indirect and direct action of the external conditions of life, and from use and disuse; a Ratio of Increase so high as to lead to a Struggle for Life, and as a consequence to Natural Selection, entailing Divergence of Character and the Extinction of less-improved forms. Thus, from the war of nature, from famine and death, the most exalted object which we are capable of conceiving, namely, the production of the higher animals, directly follows. There is grandeur in this view of life, with its several powers, having been originally breathed into a few forms or into one; and that, whilst this planet has gone cycling on according to the fixed laws of gravity, from so simple a beginning

endless forms most beautiful and most wonderful have been, and are being, evolved.

- Charles Darwin, *The Origin of Species* (1859)



Carl Buell.

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Preface

FEW SOUNDS ARE AS JOYFUL for authors as the thud made by a mail carrier dropping the first printed copy of their book by the front door. Years of effort suddenly take shape as paper, ink, and covers. But for textbook authors, there is another sound that's just about as sweet: receiving the first printed copy of the second edition of a textbook.

By the time a trade book about science reaches the bookstore, it's probably already out of date. Science rolls forward, as researchers perform surprising new experiments or stumble across fossils lurking in the ground. Coaxing a trade publisher to update a book is next to impossible, for a whole host of economic reasons. Fortunately, textbook publishers think otherwise. A new edition of a textbook can be an update on a scientific field, rather than a cultural artifact. It is thus with great pleasure that I present the second edition of *The Tangled Bank: An Introduction to Evolution*.

The first edition, published in 2009, was an experiment of sorts. A growing number of colleges were offering evolution-related courses to nonbiology majors, but they had no textbook specifically tailored for their students. Having spent two decades reporting on evolutionary biology for newspapers and magazines, as well as writing trade books on the topic, I decided to create just such a textbook.

The response has been immensely gratifying, and it's encouraged me as I've revised *The Tangled Bank* for a new edition. Much of the work I've put into this edition has involved surveying recent scientific literature for new studies that advance our understanding of evolution. Those advances have included everything from research on our Neanderthal relatives to experiments on cancer-fighting drugs based on evolutionary principles.

The second edition contains new figures that better illuminate the concepts I discuss in the text. I've also updated the overall organization. In response to

a number of requests, for example, I've written an entirely new chapter near the end of the book dedicated to human evolution. There I integrate the concepts introduced earlier in *The Tangled Bank*, such as phylogeny, genetic drift, and sexual selection, to help readers gain a deeper understanding of our own species.

From the start of this project, I've tried to make The Tangled Bank as readable as possible. The second edition does not have some of the standard pedagogical conventions. New terms are not set off in boldface, for example, and the book does not contain problem sets or summaries. Instead, I define new terms in the text itself. Students who want to check their understanding can now consult a new study guide, produced by biologist and journalist Alison Perkins. For more information on purchasing the study guide, visit http://www.roberts-publishers.com.

Although the second edition of *The Tangled Bank* is new in some important ways, it does not waver from the goals of the first edition. I hope that it continues to help a wide range of students come to appreciate the majesty and explanatory power of evolution.

— CARL ZIMMER, GUILFORD CT MAY 2013

SECOND EDITION THE TANGLED BANK

Walking Whales

Introducing Evolution



Carl Buell.

The earliest whales, such as the 47-million-year-old *Ambulocetus*, still had legs. Their anatomy gives us clues to how whales made the transition from land to sea.

One of the best feelings paleontologists can ever have is to realize that they've just found a fossil that will fill an empty space in our understanding of the history of life. Hans Thewissen got to enjoy that feeling one day in 1993, as he dug a 47-million-year-old fossil out of a hillside in Pakistan. As he picked away the rocks surrounding the bones of a strange mammal, he suddenly realized what he had found: a whale with legs.

A hundred million years ago, not a single whale swam in all the world's oceans. Whales did not yet exist, but their ancestors did. At the time, they were small, furry mammals that walked on land. Over millions of years, some of the descendants of those ancestors underwent a mind-boggling transformation. They lost their legs, traded their nostrils for a blowhole, and became creatures of the sea. This profound change was the result of evolution.



Photo by J.G.M. Thewissen, NEOMED.

Paleontologist Hans Thewissen has discovered many of the bones of *Ambulocetus* in Pakistan.

This book is an introduction to that process. Over the course of some 3.5 billion years, life has evolved from humble beginnings into the tremendous diversity found today on Earth—from whales to tulips to viruses. Indeed, to understand life on Earth—its diversity of forms, of biochemistry, of behaviors—it's crucial to understand evolution. The great twentieth-century biologist Theodosius Dobzhansky summed up the importance of this branch of science in 1971 when he declared, "Nothing in life makes sense except in the light of evolution" (Dobzhansky 1973).

The Tangled Bank is intended for anyone with a curiosity about how life works. To explain mathematical concepts, for example, it uses graphs and diagrams rather than detailed equations. This book presents the research of scientists like Thewissen not in technical detail, but through their own experiences of discovery. When Charles Darwin formulated the theory of evolution in the mid-1800s, the most sophisticated tool he could use was a crude light microscope. As we'll see in this book, scientists today can study evolution in many ways—by analyzing our DNA, for example, by probing the atoms of ancient rocks to determine the age of fossils, or by programming powerful computers to decipher how a thousand different species are related to each other.

Understanding evolution is too important to be limited only to evolutionary biologists. Evolution underlies many important issues society faces, and a better understanding of evolution can help us to grapple with them. The world's biodiversity faces many threats, from deforestation to global warming. By studying past mass extinctions, we can better understand our current crisis. Evolution has produced bacteria that can resist even the most powerful antibiotics. Scientists can observe this resistance as it evolves in

laboratory experiments. The evolution of pests and pathogens poses a serious risk to our food supply. But evolution can also help us to address some of the biggest questions we ask about the universe and ourselves: How did we get here? What does it mean to be human?

What Is Evolution?

The short answer is *descent with modification*. The patterns of this modification, and the mechanisms by which it unfolds, are what evolutionary biologists study. They have much left to learn—but at this point in the history of science, there is no doubt that life has evolved and is still evolving.

The fundamental principles that make evolution possible are pretty straightforward. Organisms inherit traits from their ancestors because they receive a molecule called DNA from them (see <u>page 115</u> for a discussion of heredity). Cells use DNA as a guide to building biological molecules, and, when organisms reproduce, they make new copies of DNA for their offspring. Living things do not replicate their DNA perfectly; sometimes sequence errors occur. Such errors are referred to as mutations (<u>Chapter 5</u>). A mutation may be lethal; it may be harmless; or it may be beneficial in some way. A beneficial mutation may help an organism to fight off diseases, to thrive in its environment, or to improve its ability to find mates.

Evolution takes place because mutated genes become more or less common within populations over the course of generations. Many mutations eventually disappear, while others spread widely. Some mutations spread simply by chance. Others spread because they allow organisms to produce more offspring. This nonrandom spread of beneficial genes is known as natural selection. The effect of a mutation depends on more than just the mutation itself. It is also influenced by all the other genes an organism carries. The environment in which an organism lives can also have a huge effect. As a result, the same mutation to the same gene may be harmful in one individual and harmless in another. Natural selection may favor a mutation in one set of circumstances and may drive it to oblivion in another.

These processes are taking place all around us every day, and they have been accumulating genetic changes ever since life began at least 3.5 billion years ago (<u>Chapter 3</u>). Darwin argued that over such vast stretches of time, natural selection could have produced complex organs, from the wings of birds to human eyes. Today, the weight of evidence overwhelmingly supports that conclusion. Changes in organs are not the only adaptations that have

emerged through evolution; behavior and even language have evolved as well (<u>Chapters 13</u> and <u>14</u>).

To trace the history of these traits, evolutionary biologists reconstruct how species diverged from a common ancestor (<u>Chapter 4</u>). Natural selection and other processes can make populations genetically distinct from one another. Over time, the populations become so different from one another that they can be considered separate species (<u>Chapter 9</u>). One way to picture this process is to think of the populations as branches on a tree. When two populations diverge, a branch splits in two. As the branches split again and again over billions of years (and sometimes came back together), the tree of life emerged.

To reconstruct the tree, evolutionary biologists identify which species are closely related to each other. They do so by comparing anatomical traits and DNA among many different species. Close relatives share more traits inherited from their common ancestor. We humans have a bony skull, for example, as do all other mammals, as well as birds, reptiles, amphibians, and fishes (<u>Chapter 4</u>). We are more closely related to these animals than we are to animals without a skull, such as earthworms and ladybugs.

Evolutionary biologists investigate not only adaptations, but the shortcomings of those adaptations. Life does not evolve by steadily progressing toward some particular goal. Our apelike ancestors did not evolve big brains because they "needed" them, for example; the conditions in which they lived—searching for food on the African savanna in big social groups—were such that some traits were favored over others (Chapter 14). Our ancestors benefited from a bigger, more complex brain, but in some ways it's a spectacularly maladapted organ. The human brain is so big that it makes childbirth much more dangerous for human mothers than for other female primates, for example. It also requires a huge amount of energy to supply—one out of every five calories you eat is used to fuel your brain.

Evolution is imperfect, because it does not invent things from scratch: it only modifies what already exists. Any organism can acquire only a limited number of beneficial mutations, and so evolution can produce new forms only under tight constraints. Because mutations can have several different effects at once, evolution also faces trade-offs. Mutations that increase the number of offspring an organism can have may also cut down its life span. These tradeoffs underlie some of the diseases that are common in the elderly,

such as cancer. As a result, studying evolution can help researchers better understand—and perhaps even treat—these diseases (<u>Chapter 15</u>).

Evolution has produced many helpful partnerships in nature, such as the one between flowering plants and the insects that pollinate them—a partnership we depend on for many of our crops (Chapter 12). But evolution does not produce a peaceful balance in the natural world. The same process species use in adapting to one another can also give rise to what looks to us like cruelty. Predators are exquisitely adapted to finding and killing prey. Parasites can devour their hosts from the inside out. Their adaptations are finely honed, allowing them to manipulate individual molecules within their hosts. Yet parasites and predators are not evil. They are just part of a dynamic balance that is constantly shifting, driving the diversification of life but also leading to extinctions.

The diversity of nature, in other words, is not eternally stable. More than 99 percent of all species that ever existed have become extinct, and, at some points in Earth's history, millions of species have disappeared over a relatively short span of time. We may be at the start of another period of mass extinctions, this time caused by our own actions (<u>Chapter 11</u>).

A Case of Evolution: Why Do Whales Have Blowholes?

Evolutionary biology is a science of integration. To investigate questions about life, evolutionary biologists integrate evidence from many different sources, gathered with many different methods. Throughout this book, I take a close look at many examples of scientists weaving together different pieces of research to understand evolution. These examples run the gamut, from venomous snakes to drug-resistant bacteria. But there's no better way to launch an exploration of evolution than by looking at whales and dolphins (FIGURE 1.2).



Left: Jo Crebbin/Shutterstock; center: Douglas Atmore/Getty Images; right: Styve Reineck/Shutterstock.

FIGURE 1.2

Living whales all share a number of traits, such as blowholes and horizontal tail flukes. Some species have filter-like growths in their mouths that they use to sieve small animals from seawater (left). Other species, such as killer whales (center) and dolphins (right), have peg-shaped teeth that they use to grab larger prey such as fish and seals.

Charles Darwin, who established the major concepts of evolution in his 1859 book *The Origin of Species*, was the first evolutionary biologist to think about how whales evolved. He recognized that living whales and dolphins (known collectively as cetaceans) are a biological puzzle. They have fish-like bodies, sculpted with the same sleek curves you can find on

tunas and sharks, so they use relatively little energy to shoot through the water. The tails of whales and dolphins narrow down to a small neck and then expand into flattened flukes, which they lift and lower to generate thrust. Sharks and tunas have similar tails, but they move theirs from side to side.

Despite their fish-like appearance and ecology, however, cetaceans are different from fishes in some profound ways. To breathe, for example, they must rise to the surface of the ocean. Then a blowhole on the top of the head opens and draws air into a passageway leading to their lungs. Most fish species can get all the oxygen they need by pumping water through their gills, where some of the dissolved oxygen passes into their blood vessels. Cetaceans have tiny bones embedded in their flesh just where the hips would be on a land vertebrate. Fishes have relatively simple sets of muscles that form vertical blocks from head to tail, whereas whales and dolphins have long muscles that run the length of their bodies—much like the long muscles running down your back. Cetaceans give birth to live young that cannot get their own food; instead, they must drink milk that their mothers produce with special mammary glands.

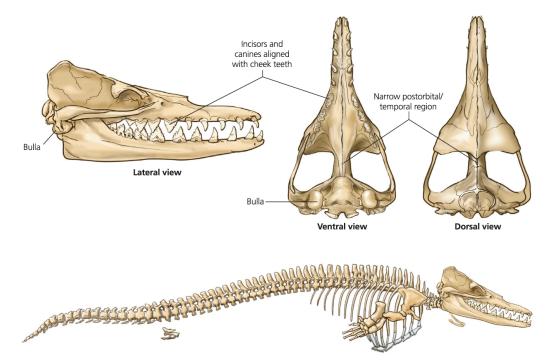
Together, these differences make cetaceans utterly unlike fishes. The only group of species where you can find such a combination of traits is mammals.

Darwin proposed a straightforward explanation for this puzzling pattern of similarities and differences. Cetaceans descended from mammals that lived on land, and their lineage evolved into marine mammals through natural selection. The ancestors of modern whales lost their hindlimbs, and their front legs became shaped like flippers. But today whales still have many traits they inherited from their mammalian ancestors, such as lungs and mammary glands.

Evolution, as Darwin envisioned it, was a gradual process. A land mammal did not give birth to a full-blown whale, in other words. If Darwin was right about this, then intermediate species of cetaceans must have existed in the past. These transitional forms would have started out being adapted to life on land and then gradually adapted to water instead. But Darwin could not point to such a fossil record, because in the mid-1800s, paleontologists were only starting to dig up extinct cetaceans. When Darwin died in 1882, he was not much wiser about how cetaceans evolved.

The first cetacean fossils paleontologists discovered were already completely adapted to life in the water. *Dorudon*, a species that lived 40

million years ago, is a typical example of these ancient cetaceans (**FIGURE 1.3**). Its flippers and long vertebral column were very much like those of living cetaceans. The most compelling evidence that *Dorudon* was a cetacean was far subtler, however. The mammalian middle ear is an air-filled cavity enclosed in a hollow shell. Whales and dolphins have this same structure, but with some distinctive features. On one edge of the shell is a thick lip of dense bone, called the involucrum. In no other group of mammals do scientists find an involucrum. *Dorudon* has an involucrum—providing strong evidence that it's a cetacean (<u>Thewissen et al. 2009</u>).



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company

FIGURE 1.3

In the late 1800s, paleontologists began to discover early whales, such as *Dorudon*, a 5-meter-long whale that lived 40 million years ago in Egypt. *Dorudon* was a completely aquatic whale and had a number of traits found today only in living whales, such as the tooth and skull features noted in this figure. The grape-shaped bone called the bulla contains bones of the middle ear; some anatomical features within the bulla of *Dorudon* are also shared by living whales. On the other hand, *Dorudon* also had some traits not found in living species, including a blowhole midway up its snout and complete hind legs (albeit very small ones). Living whales have cone-shaped teeth, while *Dorudon* had more

complex ones that resemble those of certain extinct land mammals. This combination of traits reflects *Dorudon*'s transitional position in the evolution of whales. (Information from <u>Uhen 2010</u>.)

In other ways, however, *Dorudon* was also different from any living cetacean. Consider its teeth. *Dorudon* had incisors in the front and molars in the back. Each tooth had a complex set of cusps and ridges. No living whale has anything like this dental arrangement. One group of cetacean species, known as odontocetes, has simple cone-shaped teeth. The other cetaceans, known as mysticetes, have no teeth at all: instead, they filter out small animals through baleen. The teeth of *Dorudon* are actually much more similar to those of land mammals than of living cetaceans. This trait could well be the result of the gradual evolution of whales: long after the rest of their body had evolved many adaptations to the water, early whales still had teeth like terrestrial mammals.

Cetacean evolution started to come into much sharper focus in the late 1970s. At the time, Philip Gingerich—a young paleontologist from the University of Michigan—was paying regular visits to Pakistan to investigate a geological formation from the Eocene period, 56 million to 34 million years ago. Gingerich wanted to document the mammals that existed in that region during that time. He and his colleagues brought back a number of fossils to Michigan and then slowly prepared them in his laboratory, extracting the fossilized bone from the surrounding rock. One 50-million-year-old fossil—the teeth and back portion of a wolf-sized skull—was particularly intriguing. It bore some striking similarities to cetaceans such as *Dorudon*, especially in its teeth. Gingerich concluded that he had found the earliest known cetacean, which he and his colleagues dubbed *Pakicetus* (Gingerich, Rose, and Krause 1980).

Gingerich's discovery was dramatic not just for the great age of the fossil but also for where he had found it. Whales and dolphins today live only in the water—most live in the ocean, and a few species of dolphins live in rivers. The rocks in which *Pakicetus* fossils formed offer clues to the environment in which it lived. Geologists have determined that those rocks formed as sediment accumulated in shallow streams that flowed only

seasonally through a hot, dry landscape (<u>Thewissen et al. 2009</u>). This evidence strongly suggests that *Pakicetus* lived on land (<u>FIGURE 1.4</u>).

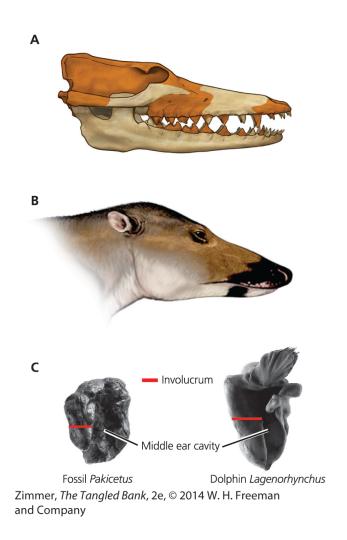


FIGURE 1.4

A: The 50-million-year-old *Pakicetus* was the first terrestrial whale ever discovered. This reconstruction is based on fossils from several different individuals. The fossil material is colored red. B: A painter's reconstruction of what *Pakicetus* looked like in life. C: *Pakicetus* bears certain hallmarks found in only cetaceans and no other mammals. On the right is a bone (from a dolphin) called the ectotympanic. It is located in the bulla, which surrounds the middle ear cavity. In living cetaceans, the inner wall of the ectotympanic forms a thick, dense lip called the involucrum. On the left is the ectotympanic from *Pakicetus*. It has a similar involucrum. Such uniquely shared traits reveal that *Pakicetus* was an ancient relative of living whales and dolphins. (Information from Cooper et al. 2009; Gingerich et al. 1983; Luo and Gingerich 1999; Nummela et al. 2006; Thewissen et al. 2009.)

Gingerich's discovery was the first of a dazzling number of ancient cetacean fossils that have come to light over the past 30 years. One of the most important was discovered by Thewissen, who studied with Gingerich in the late 1980s. As part of his graduate school research, Thewissen traveled to a different part of Pakistan to look for mammals that lived a few million years after *Pakicetus*. One day Thewissen and his Pakistani colleagues happened across a strange fossil of a large mammal. They slowly excavated its bones from the tail to the head. Its tail was massive, its legs were stubby, and its rear feet were shaped like paddles. Even as Thewissen was digging up the fossil, he could see that its head was long like an alligator's, but it had teeth like those of fossil whales (FIGURE 1.5).



Photo by J.G.M. Thewissen, NEOMED.

FIGURE 1.5

The skeleton of *Ambulocetus* shares the hallmarks found today only in whales, such as an involucrum. It was also the first fossil whale ever discovered with complete legs. A reconstruction of Ambulocetus is shown on <u>page 2</u>.

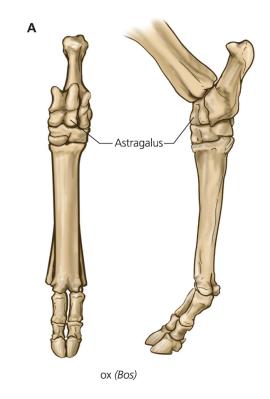
Thewissen brought the fossil to the United States, where he continued preparing and analyzing it. He discovered more traits linking it to cetaceans, including a thickened involucrum. He concluded that the fossil was, in fact, a whale that could walk. He named it *Ambulocetus:* "walking whale"

(<u>Thewissen et al. 1997</u>; <u>Zimmer 1998</u>). A reconstruction of *Ambulocetus* can be found on the opening page of this chapter.

In the years that followed, paleontologists found many fossils of different species of whales with legs. Thewissen, who now teaches at Northeast Ohio Medical University, has found additional bones of the older cetacean ancestor *Pakicetus*. Fragments of its skull revealed it to have a cetacean-like involucrum and also showed that its eyes would have sat on top of its head (see <u>Figure 1.4</u>). Fossil bones from other parts of its skeleton have led Thewissen to reconstruct *Pakicetus* as a wolf-sized mammal with slender legs and a pointed snout. Gingerich, meanwhile, found the fossils of another cetacean called *Rodhocetus*, whose short limbs resembled those of a seal.

As the evidence that cetaceans evolved from land mammals grew, scientists wondered which group of land mammals in particular they had evolved from. One way to address this question is to compare the DNA in living species. As we'll see in Chapter 7, scientists can use genetic information to draw evolutionary trees showing the relationships between species. In the 1990s, several research groups began comparing snippets of DNA from cetaceans and other mammals. They concluded that those species were most closely related to a group of mammals called artiodactyls, which includes cows, goats, camels, and hippos. As they compared more DNA from these species, the cetacean-artiodactyl link only strengthened. In fact, the scientists concluded that cetaceans were most closely related to hippos (Price, Bininda-Emonds, and Gittleman 2005).

This conclusion was a hypothesis, and like all good hypotheses, it could be tested. All living and fossil artiodactyls, for example, share a distinctive ankle bone called an astragalus. Unlike any other mammal, artiodactyls have an astragalus with an end shaped like a pulley (FIGURE 1.6). If cetaceans did indeed evolve from an artiodactyl, then the early cetaceans that still had feet should bear a double-pulley astragalus. As it happens, this small bone is rarely preserved in a fossil. But Thewissen and Gingerich both discovered the bone on ancient cetaceans—and, in both cases, it was indeed pulley shaped.





Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo by J.G.M Thewissen, NE OMED.

FIGURE 1.6

A: Artiodacty is are a lineage of mammals that includes cows, hippos, and giraffes. All artiodacty is have an ankle bone, called the astragalus, with a distinctive double-pulley shape. B: The astragali of two living artiodacty is are shown here next to the fossil astragali of two ancient whales. The double-pulley shape of these bones is evidence that whales evolved from artiodacty is on land (Thewissen et al. 2009).

To see how cetaceans evolved from artiodactyls, it helps to draw the evolutionary relationships between species. As shown in FIGURE 1.7, such an image can take the form of a tree. Each line in the tree represents a lineage reproducing through time; lineages split to produce new ones. We have some clues about the age of these lineages from different kinds of evidence, such as fossils. The thick lines in Figure 1.7 show lineages that are documented in the fossil record; the thin lines, the lineages we can infer from the evidence. (In Chapter 4, we'll explore these trees and how to interpret them in more detail.)

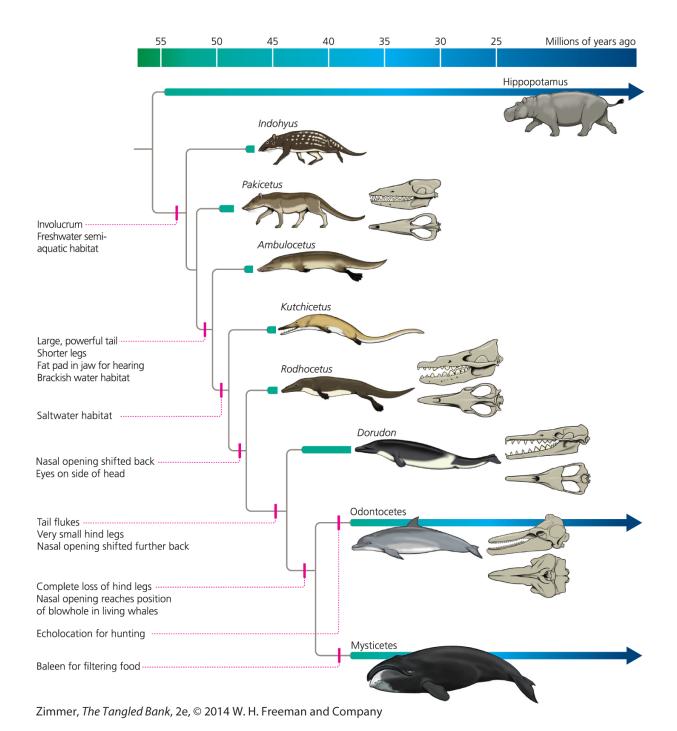


FIGURE 1.7

The relationship between some extinct species of early whales and living species. The animals illustrated here are only a fraction of the fossil whales that paleontologists have discovered in recent decades. By studying fossils, paleontologists have been able to show how the traits found in living whales evolved gradually rather than all at once.

The earliest cetaceans, such as *Indohyus* and *Pakicetus*, had four legs. They may have been able to swim, but only by kicking their legs like many land mammals—like hippos—do today. From such terrestrial ancestors, other lineages evolved that were more adapted to life in water. *Ambulocetus* lived about 49 million years ago along the coastline of what is now Pakistan. It had short legs and massive feet. Its skeleton suggests that it swam like an otter, kicking its large feet and bending its tail. *Rodhocetus*, with its seal-like limbs, probably could only drag itself around on land.

The pattern of evolution in the fossil record suggests that natural selection favored adaptations, such as short legs, that made cetaceans more efficient at swimming in water. But evolution did not completely retool cetaceans from the ground up. Cetaceans did not evolve gills, for example. Instead, the phylogeny of cetaceans indicates that their nostrils gradually shifted up to the top of their skulls, allowing them to take in air more efficiently each time they rose to the surface of the water.

Cetacean fossils also show that the loss of hind legs took millions of years. Some 40 million years ago, fully aquatic species such as *Dorudon* had evolved. But *Dorudon* still had small but complete hindlimbs. (A related cetacean of the same age, called *Basilosaurus*, provides an even more spectacular demonstration of this evolutionary lag: it grew to the size of a school bus, and yet had hind legs, complete with ankles and toes, the size of a young child's.)

The embryos of living cetaceans offer some clues about how cetaceans lost their legs. Legs arise in mammal embryos as a distinctive set of genes become active at one end of the embryo. Hans Thewissen, collaborating with a team of embryologists, discovered that these same genes also become active in dolphin embryos. Tiny buds of tissue sprout from the flanks of the dolphin embryo as they do in other mammals (FIGURE 1.8). But these limb buds stop growing after a few weeks and then die back. The study by Thewissen and his colleagues points to a compelling hypothesis for how whales lost their legs: mutations arose that could stop the growth of the limbs shortly after they had started forming (Thewissen et al. 2006). In Chapter 8, we'll see how this kind of evolutionary change to development has helped produce life's diversity.

Weeks 4-9 of embryonic development



Zimmer, The Tangled Bank, 2e, © 2014 W. H. Freeman and Company

FIGURE 1.8

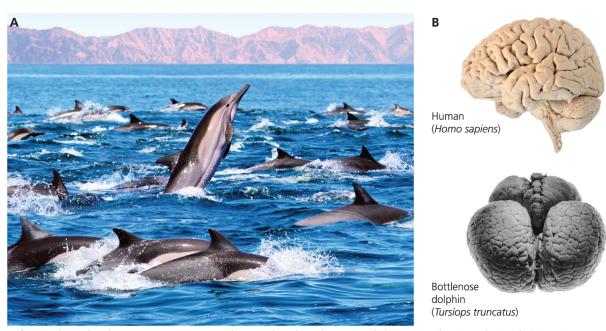
A dolphin embryo grows buds of both forelimbs and hindlimbs. The forelimb buds go on to develop into fins. The hindlimbs, on the other hand, are absorbed back into the embryo. Scientists have found that simple changes in the timing of gene expression have helped transform early terrestrial whales into their fish-like descendants (<u>Thewissen et al. 2009</u>).

The early cetaceans evolved into many lineages, most of them long extinct. The odontocetes and mysticetes, the two lineages of cetaceans still in existence today, split from a common ancestor about 40 million years ago. After that split, the odontocetes evolved muscles and special organs that they used to produce high-pitched sounds in their nasal passages leading to their blowholes. Listening to the echoes that bounced off objects around them in the water, they could perceive their surroundings. Today, dolphins and other odontocetes use these echoes to hunt for their prey. The mysticetes followed a different trajectory: They didn't evolve echolocation. Instead, they lost their teeth and evolved huge, stiff pleats in their mouths that allowed them to filter prey from huge volumes of water.

Scientists are now beginning to find important new clues to the origins of both groups. Fossils have revealed that the earliest mysticetes still had teeth, for example, and probably grew only small patches of baleen at first (<u>Uhen 2010</u>). Only later did their teeth disappear. Mysticetes still carry genes for building teeth, but most of them have acquired disabling mutations.

Biologists have long been impressed with the size and complexity of whale brains (<u>FIGURE 1.9</u>). Aside from humans, dolphins have the biggest brains in proportion to their bodies of any animal (<u>Marino 2007</u>). Dolphins

can also use their oversized brains to solve remarkably complicated puzzles that scientists make for them. A number of studies suggest that big brains evolved in dolphins as a way to solve a particular kind of natural puzzle: figuring out how to thrive in a large social group. Dozens of dolphins live together, forming alliances, competing for mates, and building relationships that persist throughout their long lives. They communicate with each other with high-frequency squeaks, and each dolphin can tell all the other dolphins apart by their whistles. Natural selection appears to have favored dolphins with extra brainpower for processing social information (Connor 2007).



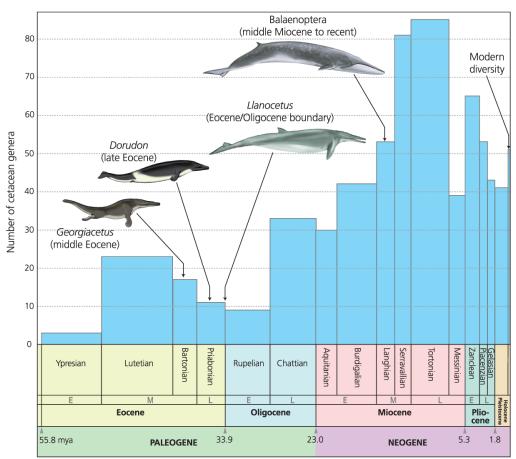
Left: David M Schrader/Getty Images; top right: Jesada Sabai/Shutterstock; bottom right: Naruaki Onishi/Getty Images.

FIGURE 1.9

A: Dolphins live in large groups and can communicate to each other with complicated sounds. B: Their complex social life may have favored the evolution of big, powerful brains. Relative to their body size, dolphin brains are second only to those of humans.

One of the big lesson of the fossil record is that groups of species rise and fall in diversity over evolutionary times (<u>Chapter 11</u>). Cetaceans are no exception. Mark Uhen, a paleontologist at George Mason University, recently tallied the diversity of cetaceans over the past 50 million years, and his research chronicles dramatic fluctuations in the number of species (<u>FIGURE</u>

1.10; <u>Uhen 2010</u>). There are many such patterns in the fossil record, and evolutionary biologists are testing hypotheses to explain them. Uhen has argued that cetacean evolution kicked off with an explosion of semiaquatic species such as *Ambulocetus*. By the end of the Eocene, however, fully aquatic forms like *Basilosaurus* had evolved, and the semiaquatic forms had become extinct (<u>Uhen 2010</u>). The fully aquatic cetaceans became top predators in the ocean ecosystem.



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FIGURE 1.10

The fossil record of whales documents increases and decreases in the diversity of species over the past 55 million years. Paleontologists have hypothesized that changes in the planet's climate and the ocean's ecosystems have driven these long-term changes (<u>Uhen 2010</u>).

The two living lineages of cetaceans evolved very different strategies for hunting: the mysticetes trapped small animals with their baleen while the odontocetes swam after fishes and larger prey. But in both cases, they depended on productivity of the ocean food web for their food. Uhen and Felix Marx of the University of Bristol have observed that cetacean diversity shot up after large, shelled algae called diatoms became diverse in the ocean starting about 20 million years ago (Marx and Uhen 2010). Uhen and Marx argue that the explosion of diatoms provided a food supply for animals that in turn were preyed upon by a diversity of cetaceans.

Unfortunately for cetaceans, a new predator has entered their lives in the past few centuries: humans. In the 1800s, sailors crisscrossed the world to hunt big whales for their oil and baleen (the oil was used for lamps and the baleen for corset stays). Many species of whales came perilously close to extinction before the whale-oil industry collapsed and laws were passed to protect the surviving animals (FIGURE 1.11).



Archive Photos/Getty Images.

FIGURE 1.11

Whale hunting in the nineteenth century nearly drove many species of whales extinct. They have been rebounding slowly, although today they continue to face many threats from human activity.

Whales reproduce slowly, and so their populations are still far from their pre-hunting levels. The small sizes of their populations put them at a greater risk of extinction. Diseases and other threats, such as pollution and heavy fishing, can destroy large fractions of small populations more readily than big ones. Small populations also have little genetic variability, making them more susceptible to genetic disorders (Chapter 6).

In the 1950s, for example, an estimated 6000 Chinese river dolphins (*Lipotes vexillifer*) lived in the Yangtze River (FIGURE 1.12). But the booming Chinese economy led to massive pollution of the river, sickening the animals. Fishermen killed many of them by accident in their nets. Because of these human activities, the Chinese river dolphin population began to crash; in 1997, an extensive search revealed only 13 individuals. And the last Chinese river dolphin was spotted in the river in 2007 (Turvey 2009). It's possible that a few dolphins are surviving somewhere along the Yangtze; but with each passing year, it is more likely that these remarkable creatures have become extinct and will never be seen again.



Avalon/Photoshot License/Alamy Stock Photo.

The Chinese river dolphin (*Lipotes vexillifer*) is believed to have become extinct in recent years due to pollution and overfishing.

Even as we humans have come to understand the remarkable history of cetaceans over the past 50 million years, their future evolution has ended up in our hands.

Most people find whales and dolphins awe-inspiring the moment they set eyes on these remarkable animals. Learning about the evolution of whales and dolphins only deepens their fascination. By probing the history of life—of cetaceans and the millions of other species we share this planet with—we can come to understand the tapestry of change that has been weaving itself for billions of years. Charles Darwin put it best with the closing lines of *The Origin of Species*: "There is a grandeur in this view of life, with its several powers, having been breathed into a few forms or into one; and that, whilst this planet has gone cycling on according to the fixed law of gravity, from so simple a beginning endless forms most beautiful and most wonderful have been, and are being evolved."

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A Brief History of Evolutionary Biology



Wayne Lynch/All Canada Photos/Superstock.

The Galápagos Islands in the Pacific are home to many species found nowhere else on Earth—such as this marine iguana. Visiting the islands in 1835 helped Charles Darwin develop his theory of evolution.

In the Pacific Ocean, seven hundred miles west of Ecuador, lies an isolated cluster of extinct volcanoes known as the Galápagos Islands. On these strange outcrops live strange kinds of animals. There are great birds with bright blue feet. There are scaly iguanas that leap into the ocean to eat seaweed and then wade back out to bask on the rocks. Giant tortoises chew peacefully on cactuses. This tiny archipelago is home to 13 species of finches found nowhere else on earth. They are so tame that they will let you hold them in your hand.

Every year, dozens of scientists come from across the world to the Galápagos islands to study these species, which exist nowhere else on earth. The islands are like a laboratory of evolution where scientists can study an isolated example of how life changes over millions of years. It takes them a long time to get to the Galápagos islands—but not as long as the journeys by steamer that scientists took a hundred years ago. And those steamer trips were much faster than the voyage of a British surveying ship that sailed to the Galápagos Islands in 1835. On board the HMS Beagle was a young British naturalist named Charles Darwin.



Iberfoto/Superstock.

Charles Darwin (1809–82) laid the foundation of modern biology with his theory of evolution.

Darwin had been traveling aboard the *Beagle* for almost four years, during which time he had studied the marine life of the Atlantic, trekked in the jungles of Brazil, and climbed the Andes. But even after all that, Darwin was astonished by the Galápagos Islands. "The natural history of this archipelago is very remarkable: it seems to be a little world within itself," he wrote later in his book *The Voyage of the Beagle* (Darwin 1845). Darwin spent five weeks on the islands, clambering over jagged volcanic rocks and gathering plants and animals. The experience would lead him over the next few years to launch a scientific revolution.

Darwin was born in 1809, at a time when most people—including the world's leading naturalists—thought the world was only thousands of years old, not billions. They generally believed that species had been specially created, either at the beginning of the world or from time to time over Earth's history. But after Darwin

returned from his voyage around the world in 1836, his experiences in places like the Galápagos Islands caused him to question those beliefs. He opened a notebook and began jotting down ideas for a new theory of life, one in which life evolved.

This was, without a doubt, a pivotal moment in the history of science. But history doesn't happen in a moment. Evolutionary biology has roots that reach back many centuries before Darwin. He was not the first person to contemplate evolution, nor did he uncover all of the evidence he used to build his argument. That evidence was the work of many generations of naturalists. Darwin reassessed that evidence, combined it with all of his own observations and inferences, and came up with a powerful new theory. Once Darwin presented his theory to the world, science did not grind to a halt. Many of the most important developments in evolutionary biology did not occur until decades after his death.

Darwin's legacy, in other words, is not some fixed set of facts. It is a way of investigating nature—a scientific method for bringing billions of years of history to light.

Nature before Darwin

Charles Darwin first began to learn about nature as a teenager in the 1820s. The concepts he was taught had emerged over the previous two centuries, as naturalists pondered two questions: what were the patterns in nature's diversity, and how did those patterns come to be?

Understanding the diversity of life had been a practical necessity. People needed names for different kinds of plants and animals, for example, so that they could pass on their wisdom about which kinds were safe to eat or useful as medicines. For thousands of years, people had been well aware that some kinds of animals and plants were similar to other kinds. Cats and cows and humans all nourished their young with milk, for example. In the 1600s, naturalists became more systematic in the way they sorted the diversity of life. They came up with rules for naming species and schemes for classifying species into different groups. This urge to compare and classify reached its most glorious form in the mid-1700s with the work of the Swedish botanist Carl Linnaeus (Koerner 1999) (FIGURE 2.2).

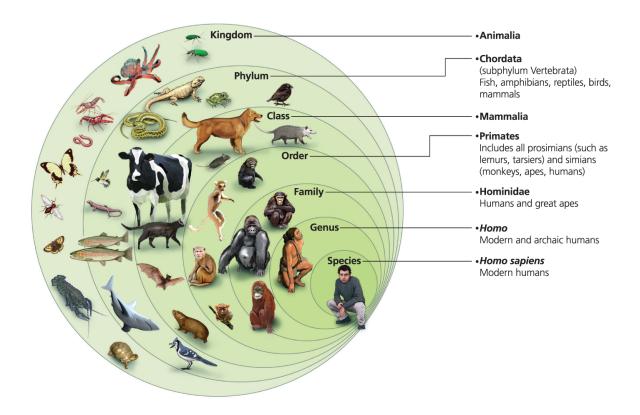


DEA/G. DAGLI ORTI/Getty Images.

FIGURE 2.2

Carl Linnaeus (1707-78) invented a system to classify species into groups.

Linnaeus organized all living things known at the time into a single hierarchy of groups. Humans belong to the mammal class, for example, and within that class, the primate order; and within that order, the family Hominidae; and within that family, the genus *Homo;* and within that genus, the species *Homo sapiens*. Linnaeus could assign every species to a particular genus, family, or order according to the traits it shared with other species. His system was so useful that biologists continue to use it today (FIGURE 2.3).



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FIGURE 2.3

Linnaeus created a system for classifying species that allowed him to nest groups of species within larger groups.

Linnaeus believed that the pattern of his system reflected a divine plan. "There are as many species as the Infinite Being produced diverse forms in the beginning," he wrote. In some cases, Linnaeus believed that species had

later changed. He held that two species of plants could sometimes interbreed, producing a new hybrid species. For the most part, though, Linnaeus believed that the overall patterns of life's diversity had not changed since the biblical creation of the world. Every species created then still existed today.

He would be proven wrong as naturalists began to study fossils. In the Middle Ages, people believed that fossils were not the remnants of living things but rather geometric forms impressed on rock, like wondrous crystals. One of the first naturalists to recognize the true nature of fossils was Nicolas Steno, a seventeenth-century Dutch anatomist and bishop in the Catholic Church. (FIGURE 2.4). In 1666, some fishermen brought him a giant shark they had caught. As Steno studied the shark's teeth, it occurred to him that they looked just like triangular rocks that were known at the time as tongue stones. Steno proposed that tongue stones had started out as teeth in living sharks. After the sharks died, their teeth gradually were transformed into stone (Cutler 2003).





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FIGURE 2.4

Nicholas Steno (1638–86) recognized that triangular rocks known as "tongue stones" were in fact fossils of teeth from sharks.

But if fossils really were the remains of once-living things, Steno would have to explain how it was that stones shaped like seashells had come to be found on top of certain mountains. How could animals that lived in the ocean end up so far from home? Steno argued that originally a sea must have covered the mountains. Shelled animals died and fell to the ocean floor, where they were covered over in sediment. As sediments accumulated, they turned to rock. The layers of rocks exposed on the sides of mountains, Steno recognized, had been laid down in succession with the oldest layers at the bottom and the youngest ones at the top.

Steno was still a traditional believer in a biblical Earth that was just a few thousand years old. Nevertheless, he was able to introduce a radically new idea: life and the planet that supported it had a history filled with change, and the Earth itself kept a record of that history.

Evolution before Darwin

The concept that life changes over the course of vast stretches of time—what came to be known as evolution—was already being vigorously debated before Darwin was born. One of the earliest evolutionary thinkers was the eighteenth-century French nobleman Georges-Louis LeClerc, Comte de Buffon (FIGURE 2.5). Buffon was the director of the King's Garden in Paris, and he owned a huge estate in Burgundy, where he harvested timber for the French navy and carried out research on the strength of different kinds of wood. Buffon also spent years writing an encyclopedia in which he intended to include everything known about the natural world (Roger 1997).



Pictorial Press LTD/Alamy Stock Photo.

FIGURE 2.5

Georges Buffon (1707–88) was one of the earliest naturalists to argue that life had changed over time.

Like other thinkers in the mid-1700s, Buffon recognized that the new sciences of physics and chemistry offered a radically different way of thinking about the universe. It had become clear that the world was made up of minuscule particles, which we now call atoms and molecules. These particles reacted with each other according to certain laws, and when they

came together into larger objects, the objects obeyed certain laws as well. They were attracted to one another by gravity, for example, and either attracted or repelled by electric charge. Following these laws, the particles moved about, and the complexity of the universe emerged spontaneously as a result.

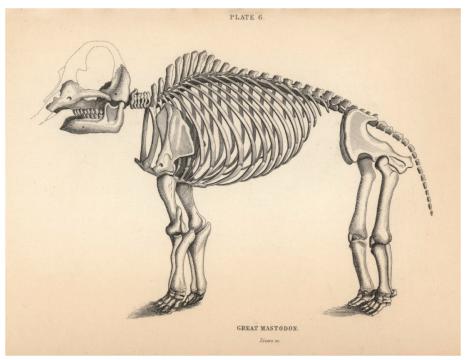
Buffon proposed that Earth had formed according to the laws of physics. A comet struck the sun, he argued, breaking off debris that formed into a planet. The scorching Earth cooled down and hardened, and oceans formed and dry land emerged. The entire process took more than 70,000 years, Buffon calculated—a span of time too vast for most people in Buffon's day to imagine.

The fact that living things were made from the same kinds of particles found in rocks and water struck Buffon as profoundly important. He argued that each species had a supply of organic particles that somehow transformed an egg or a seed into its adult form. He envisioned that these organic particles had first come together in the hot oceans of the early Earth. Animals and plants sprang into existence in the process, and, as the planet cooled, they retreated to the warm tropics. Those migrations could explain the stunning discovery in the mid-1700s of fossil elephants in Siberia and North America, far from the tropics where elephants live today.

When life first emerged, Buffon proposed, it was already divided into a number of distinct types. Each "internal mould," as he called it, organized the organic particles that made up any individual creature. But life could also be transformed. As a species moved to a new habitat, its organic particles changed, and its mould changed as well. Buffon was proposing something akin to one of the central tenets of modern evolutionary theory: populations change over time.

Fossils and Extinctions

Steno's realization that fossils were the remains of living things helped open up a new science that came to be known as paleontology. Some of the most startling discoveries were of fossils of species that no longer lived at the sites where they were unearthed—such as the Siberian elephants (Rudwick 1985). When Georges Cuvier, a French paleontologist (1769–1832), compared the elephant fossils to the skeletons of living elephants from Africa and India, he discovered that some of the fossils were distinct from living elephants in some crucial ways, such as the shapes of their teeth (FIGURE 2.6). These fossil animals, which he called mammoths and mastodons, were species that no longer existed. They had, in other words, become extinct (Rudwick 1997).



Florilegius/Getty Images.

FIGURE 2.6

In the late 1700s, paleontologists recognized that some fossils belonged to species that no longer exist, such as this mastodon, a relative of elephants that became extinct 11,000

Cuvier and others went on to document the extinction of many species. Paleontologists began to investigate how these species could have died out. The answer turned out to be hidden in the rocks themselves—or, more precisely, their geography. During the eighteenth century, a debate raged about whether Earth's features had formed from volcanic eruptions or floods. An important step forward came when James Hutton, a Scottish physician and farmer, realized that rocks formed through imperceptibly slow changes—many of which we can see around us today. Rain erodes mountains while molten rock pushes up to create new mountains. The eroded sediments form into layers of rock that can later be lifted above sea level, tilted by the force of the uprising rock, and eroded away again. Some of these changes can be tiny; but over enough time, Hutton argued, they could produce vast changes. Earth must therefore be vastly old—Hutton envisioned it as a sort of perpetual-motion machine passing through regular cycles of destruction and rebuilding that made the planet suitable for humankind.

Hutton's vision of a slowly transformed Earth came to be accepted by most geologists in the 1800s. They looked closely at layers of exposed rock and began to determine how they were formed by volcanoes and deposits of sediments. And they also began to figure out in what order those layers had formed. Some of the most important clues to the history of Earth came from fossils. William Smith, a British canal surveyor, came to this realization as he inspected rocks around England to decide where to dig canals (FIGURE 2.7). He noticed that the same kinds of fossils tended to appear in older rocks, but different ones appeared in younger layers. Smith could find the same sets of fossils in rocks separated by hundreds of miles (Winchester 2001).



Paul D. Stewart/Science Source.

William Smith (1769–1839) discovered that layers of rocks contain distinctive groups of fossils.

By the early 1800s, then, geologists came to agree that the surface of the planet had been gradually sculpted over vast spans of time. Smith realized that each type of animal had lived across a wide geographical range for a certain period of time, and so the rocks that formed during that time preserved their fossils. As those animals became extinct and new ones emerged, younger rocks contained their own sets of fossils. By marking the places where he found certain fossils, Smith was able to organize strata into a geological history, from oldest to youngest (FIGURE 2.8).

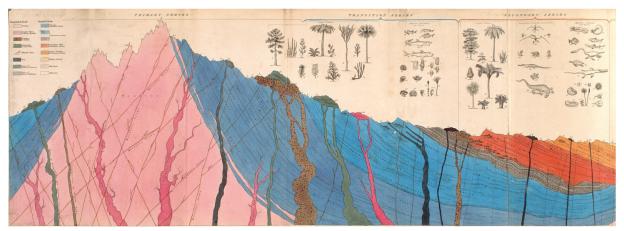


The Trustees of the Natural History Museum, London.

William Smith produced the first detailed geological map. The colors represent geological formations that formed at different times. Each formation contained a distinctive assemblage of fossils.

Other researchers, including Cuvier, later used the same method to map the geology of other parts of the world. They discovered that formations of rock exposed in one country could often be found in others. They began giving names to the sequences of these far-flung rock formations (see the geological chart on the endpapers). Many fossil species were restricted to just a few layers of rock. Larger groups of species spanned more geological history, but they had their own beginnings and endings as well. In the early 1800s, for example, fossil hunters discovered the bones of gigantic reptiles; some of them had lived on land and some in the sea. These fossils came only from

rocks dating back to the Mesozoic era and disappeared abruptly at its end (FIGURE 2.9).



The Trustees of the Natural History Museum, London.

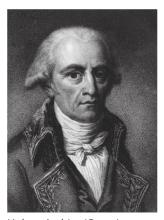
FIGURE 2.9

Early geologists organized the surface features of Earth into strata based on where certain fossils were found. This early engraving of a mountain depicts strata and the associated fossils.

Why species emerged and disappeared over the history of life was a subject of fierce debate. Cuvier, for example, rejected Buffon's earlier suggestion that life had evolved. He believed that life's history had been punctuated by revolutions that had wiped out many species and brought many new ones to take their place. But one of his colleagues at the National Museum of Natural History in Paris was about to make a new case for evolution.

Evolution as Striving

Cuvier's colleague, Jean-Baptiste Pierre Antoine de Monet, Chevalier de Lamarck, was an expert on plants and invertebrates (FIGURE 2.10). He was struck by the similarities between some of the species he studied. He was also impressed by the fossil record, which at the time was becoming detailed enough to reveal a dynamic history of life (Burkhardt 1977). Lamarck combined these two lines of thought into a single argument. He proposed that life was driven inexorably from simplicity to complexity and that humans and other large species descended from microbes. To explain why there are microbes today, Lamarck argued that primitive life was being spontaneously generated all the time. Today's bacteria are just the newest arrivals.



Hulton Archive/Getty Images.

FIGURE 2.10

Jean-Baptiste Lamarck (1744–1829) argued that complex species had evolved from simple ones.

Lamarck also believed animals and plants could adapt to their environment. If an animal began to use an organ more than its ancestors had, the organ would change during its lifetime. If a giraffe stretched its neck for leaves, for example, a "nervous fluid" would flow into its neck and make it longer. Lamarck claimed that these changes could be passed down from an animal to its offspring. A giraffe could inherit a longer neck; if it continued stretching for leaves, it would pass on an even longer neck to its descendants.

Lamarck's theory gained attention across Europe for its shocking notion that species were not fixed. But his fellow naturalists, led by Cuvier, examined the theory and found it wanting. Lamarck did not accept extinction, arguing instead that fossil species had evolved seamlessly into living forms. But in the early 1800s, Cuvier continued to amass more and more evidence that extinctions were a major feature of the history of life. Lamarck also envisioned life evolving up a seamless scale of nature, from simple to complex. But when Cuvier undertook an ambitious comparative study of major groups of animals, he concluded that they were divided by huge gulfs with no intermediates to join them. His ideas rejected, Lamarck died in poverty and obscurity in 1829 (Appel 1987).

But just eight years after Lamarck's death, a young British naturalist, newly returned from a voyage around the world, quietly embraced the notion that life had evolved. And three decades after Lamarck's death, that naturalist—Charles Darwin—would publish *The Origin of Species* and change the science of biology forever.

The Unofficial Naturalist

Today the name Darwin is practically synonymous with evolution, but he was hardly the first naturalist to wonder about nature's patterns. By the time Darwin was born in 1809, Lamarck was already famous (and infamous) for arguing that life had changed over a long history. When Darwin finally presented his own theory of evolution at age 50, however, he could not be so easily dismissed. He had assembled a towering edifice of evidence and argument for evolution.

Darwin's biography makes his breakthrough all the more remarkable. He was born into comfortable wealth, thanks to the fortune his mother's family made manufacturing china and pottery. Darwin's father, a physician, expected Charles and his brother Erasmus to follow him into medicine, and he sent them to Edinburgh for training. There Charles also learned about geology, chemistry, and natural history, and he soon realized that he would much rather spend his life studying nature than practicing medicine. It was common in Darwin's day for well-to-do young men interested in nature to train in theology and become clergymen, using their spare time to pursue their investigations. Darwin started down that path, leaving Edinburgh to study theology at the University of Cambridge.

But Darwin grew restless. He reveled in a journey to Wales, where he was able to study geological formations. He devoured books about the travels of great naturalists to distant tropical countries. And then, at age 22, Darwin got a chance to go on a voyage of his own.

In 1831, Darwin was invited to join the company of a small British navy ship, HMS *Beagle*, on its voyage around the world (FIGURE 2.11). The captain, Robert Fitzroy, was haunted by a family history of mental illness and suicide. He decided to find a gentleman who might act as an unofficial naturalist for the voyage and whose companionship might keep him from succumbing to depression. He eventually settled on the 22-year-old Darwin. Darwin was thrilled by the opportunity to explore the world—he would not return home for five years.



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Charles Darwin spent five years aboard HMS *Beagle*, traveling the world and gathering clues that he would later use to develop his theory of evolution.

The *Beagle* traveled from England to South America. Along the way, Darwin gathered fossils of extinct mammals. He trapped birds and collected barnacles. He observed the ecological complexity of the jungles of Brazil. Darwin also learned a great deal about geology in South America. He recognized the layers of rock that had gradually formed and were then reworked into mountains and valleys. He experienced an earthquake in Chile, and he observed that the shoreline had been lifted a few centimeters as a result. When Darwin set out on the *Beagle*, one of the books he brought with him was the first volume of *The Principles of Geology* (1830–33), which the Scottish lawyer and scholar Charles Lyell had just published. Lyell made the provocative argument that Earth's landscapes had been created not by gigantic catastrophes, but by a series of many small changes. During the earthquake in Chile, Darwin saw firsthand one of these changes take place as the coastline rose a small amount. Darwin's travels turned him into a passionate "Lyellian."

Darwin did not realize the full importance of his observations until he returned to England in 1836. At the Galápagos Islands, for example, Darwin had collected a number of birds that had dramatically different beaks. Some had massive beaks good for crushing seeds while others had slender, needlelike beaks for feeding on cactus plants. Darwin assumed he had found species of blackbirds, wrens, and finches. But when he gave his birds to a London ornithologist named John Gould, Gould made a surprising discovery: the birds were all finches. Despite their radically different beaks, they shared a number of telltale traits found only in finches (FIGURE 2.12).



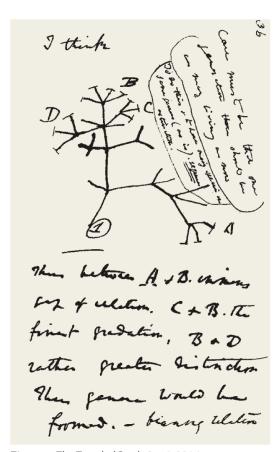
Top Left: Michael Stubblefield/Getty Images; Top Center: Michael Stubblefield/Getty Images; Top Right: iStock/Getty Images; Bottom Left: iStock/Getty Images; Bottom Center: NHPA/Superstock; Bottom Right: Mary Plage/Oxford Scientific/Getty Images.

FIGURE 2.12

Darwin was surprised to discover that finches on the Galápagos Islands have dramatically different beaks.

Darwin was puzzled. Some naturalists of his day argued that species had been created where they now were found, well suited to their climate. But if all the finches had been created on the Galápagos Islands, why were they so different from one another? Perhaps they had evolved into their current forms.

Darwin's finches helped him to conclude that all of life had evolved. Only evolution—the fact that all living things share a common ancestry—could explain the patterns in nature today. As for how life evolved, Darwin rejected mechanisms previous naturalists had proposed, such as Lamarck's ladder of progress. Instead, Darwin envisioned a process that required fewer assumptions—a process based on variation and selection (FIGURE 2.13).



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FIGURE 2.13

After returning to England, Darwin began to develop his theory of evolution by using notebooks. He drew this tree in 1837 to illustrate how different lineages evolve from a common ancestor.

Although Darwin had the basic pieces of his theory in place by the late 1830s, it would be another 20 years before he finally presented it in full detail to the public. During that time, Darwin became a highly respected researcher, known for his geological research and a massive monograph on barnacles. He came to be good friends with England's greatest naturalists, including his great inspiration, Charles Lyell. But Darwin also knew that Lyell and others had a very low opinion of Lamarck and other naturalists who had promoted evolution. And so he painstakingly worked through every possible objection that Lyell and others might raise about his own theory. Finally, in 1858, Darwin was spurred to publish his ideas when he received a letter from Indonesia.

The letter was from another English naturalist named Alfred Russel Wallace. Wallace, 14 years Darwin's junior, had patterned his own life after Darwin's famous travels. He had spent years in the jungles of South America and Indonesia, gathering plants and animals that he sold to museums and wealthy collectors in Europe. Wallace also kept careful records of the diversity of life he saw; and, as he reflected on his observations, he concluded that life had indeed evolved. He even came up with a mechanism for evolution very much like Darwin's idea of natural selection. Wallace wrote to Darwin to describe his new ideas, and he asked Darwin to present them to the Linnean Society, one of England's leading scientific organizations.

If Wallace were to publish first, Darwin knew, his own years of work could be cast into shadow. Darwin also knew that he had worked out his own argument in far more detail than Wallace. On the advice of Lyell and others, Darwin decided to turn the matter over to the Linnean Society. In July 1858, letters from both Wallace and Darwin were read at a meeting of the Linnean Society and later published in the society's scientific journal.

Strangely, though, neither the letters nor the article made much of an impression. It was not until Darwin wrote a book about his theory and published it in 1859 that the world sat up and took notice.

On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life was an immediate sensation, both in scientific circles and among the public at large. Some scientists immediately embraced "Darwinism," engaging in fierce public debates with those who rejected it. Darwin himself, however, did not

personally enter the fray. He went on working quietly and patiently at his rural home, known as Down House. There he continued to carry out experiments to investigate his theory, studying everything from orchids to earthworms. Darwin went on to write more books about evolution and other aspects of nature—including human nature, which was the subject of his 1871 book *The Descent of Man, and Selection in Relation to Sex*.

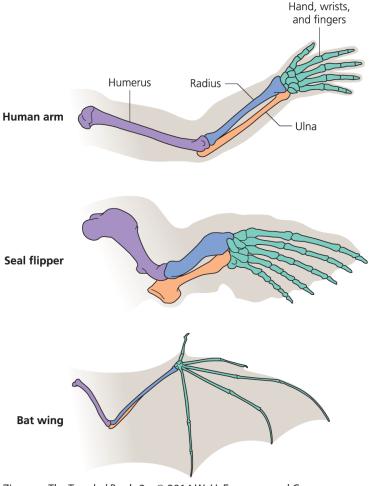
When Darwin first argued for evolution, he shocked many readers. But, over the years, much of the public came to accept a good deal of what he had to say. When *The Descent of Man* came out 12 years after *The Origin of Species*, it generated far less controversy. As the botanist Joseph Hooker, Charles Darwin's friend, wrote in a letter, "I dined out three days last week, and at every table heard evolution talked about as accepted fact, and the descent of man with calmness."

As for scientists, some were skeptical of Darwin's proposal regarding how evolution had occurred, but few disagreed that life had indeed evolved. Perhaps most important, Darwin had established evolution as a subject that could be studied scientifically: by running experiments, by comparing species, and by thinking of processes that could explain the patterns of nature. When Darwin died in 1882, he was buried in Westminster Abbey, in the company of kings and queens, great writers and prime ministers, and other great scientists including Isaac Newton.

Common Descent

One of the great achievements in *The Origin of Species* was showing the world that all species on Earth are related, like cousins in a family tree. For evidence, Darwin used the patterns of nature that had puzzled naturalists for so long.

In the mid-1800s, anatomists became keenly aware that the diversity of life had many common themes. Consider a seal's flippers, a bat's wings, and your arms (FIGURE 2.14). The seal uses its flippers to swim through the ocean; the bat uses its wings to fly; and people use their arms to cook, sew, write, perform surgery, and drive cars. These appendages serve very different functions, and yet they have a deep similarity. The bones, for example, are arranged in the same way. A long bone (the humerus) extends from the shoulder. On its far end, it meets two thin, parallel bones (the radius and ulna), and these bones can bend at the elbow. At the end of the radius and ulna is a cluster of wrist bones. The same set of bones can be found in each species' wrist. Extending from the wrist are five digits. Of course, any given bone in one species is somewhat different from the corresponding bone in other species. A seal's humerus is short and stout, for example, while a bat's looks more like a chopstick. But those differences don't obscure the arrangement that all of those limbs share. Naturalists called this similarity homology.



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Bats, humans, and seals have seemingly different limbs, which they use for different functions. But the bones in one species correspond to bones in the others. Darwin argued that this similarity was a sign of common ancestry.

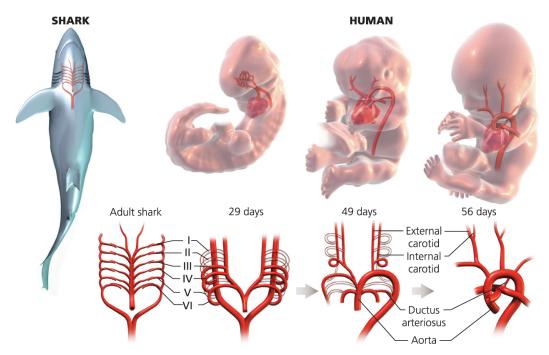
What accounts for this combination of differences and similarities? Some anatomists in the mid-1800s argued that each species was created according to an "archetype"—a fundamental plan to which some variations could be added. Darwin preferred a simpler explanation: seals, bats, and humans all shared a common ancestor that had limbs with wrists and digits. That ancestor gave rise to many lineages. In each of them the limbs evolved adaptations for different kinds of movements, such as flying, digging, and running. And yet the underlying legacy of our common ancestor survived.

Darwin's case for descent with modification was strengthened because many homologies are found together in the same groups of species. Bats, humans, and seals don't just share limbs, for example. They also have hair, and the females of each species secrete milk to nurture their young.

Taxonomists used these common traits to classify humans, bats, and seals into the same category: all three species are mammals. Darwin argued that the very fact that we can classify species this way is consistent with the notion that they evolved from a common ancestor. While new traits can evolve in different lineages (we can't fly like bats can, for example), each species descends from an ancestral mammal. And that mammal, Darwin argued, shared an even older ancestry with other animals. For example, we humans share many homologies with fish. We have eyes with the same arrangement of lenses, retinas, and nerves. We have skulls, livers, and many other organs in common.

Of course, we are different in some important ways. Just about all vertebrates on land have lungs, except for some species of lungless salamanders. Vertebrates that have returned to the ocean, such as whales and seals, haven't lost their lungs. Some fishes have lung-like structures for breathing. But they all have gills, which let them draw in dissolved oxygen from water. No land vertebrate has true gills.

Darwin argued that these differences might not actually be as profound as they first appear. In some cases, homologies are clear only when animals are still embryos, not when they are adults. Although fish and land vertebrates are still embryos, for example, they all develop the same set of arches near their heads. In fishes, those arches go on to become gills. In land vertebrates like us, they go on to form a number of different structures in the head and neck, including the lower jaw. A human embryo initially develops blood vessels in the same pattern seen in fish gills. But later, the blood vessels are modified (FIGURE 2.15). Darwin argued that those arches are homologies inherited from a common ancestor. In our ancestors, the arches that once supported gills evolved to take on a new function in adulthood.



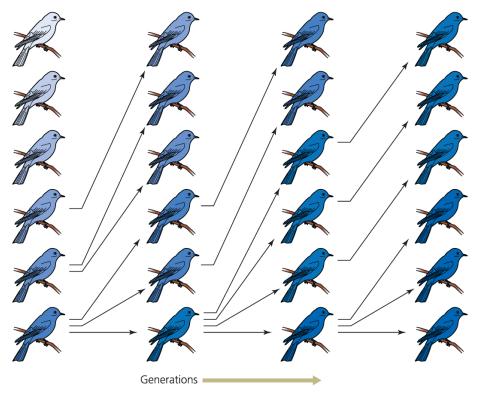
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Fishes have a series of branching blood vessels to absorb oxygen in their gills. Human embryos grow blood vessels at 29 days in the same arrangement. The vessels later change to allow us to absorb oxygen through our lungs.

Natural Selection

Darwin argued that patterns in biology—the homologies, the fossil record, and so on—could be explained by the inheritance of these features from common ancestors: in other words, evolution. He also argued for a new kind of mechanism that drove much of that change. To account for evolution, Darwin's predecessors usually proposed mysterious, long-term drives. Lamarck, for example, claimed that the history of life followed a trend toward "higher" forms. Many German biologists in the early 1800s argued that life evolved much as an embryo develops in the womb, from simple to complex. What made the new ideas of Darwin and Wallace so important? They depended on processes that were not just natural but also observable. One of the most important of these processes is natural selection.

Darwin and Wallace both found inspiration for the idea of natural selection in the writings of an English clergyman named Thomas Malthus. In 1798, Malthus published a book called *An Essay on the Principle of Population*, in which he warned that most policies designed to help the poor were doomed because population growth would always outstrip the ability of a nation to produce more food for its people. A nation could easily double its population in a few decades, but its food production would increase far more slowly. The result would be famine and misery for all. Malthus claimed that only those who could adapt to society's needs to produce useful work would be able to survive and reproduce.



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Natural selection occurs because some individuals in a species are better adapted to their environment than others. Thanks to their survival, they can produce more offspring. Over generations, their traits become more common. In this diagram, dark birds are better adapted to avoiding predators than light ones.

When Darwin and Wallace read Malthus, they both realized that animals and plants should experience just this sort of pressure. A fly can take just a few weeks to go from egg to maturity, which meant that its population could explode far more quickly than our own. But the world is not buried in a thick layer of flies. No species actually manages to reproduce to its full potential. Many individuals die before they become adults. They are vulnerable to droughts and cold winters and other environmental assaults, and their food supply is not infinite. Individuals must compete—albeit unconsciously—for the limited resources necessary for survival.

So survival and reproduction do not come down to pure chance. If an individual animal or plant has some trait that helps it to thrive in its

environment, it may leave more offspring behind than other individuals of its species, and those offspring are more likely to have that trait. That trait would therefore become more common over the course of generations.

As Darwin wrestled with natural selection, he spent a great deal of time with pigeon breeders, learning their methods. To produce new breeds—pigeons with ruffles of feathers around the neck, for example, or brilliant white plumage—breeders would select a few birds from each generation with the traits they desired. Over many generations, this selective breeding would cause the traits to get more and more exaggerated.

Darwin recognized similarities between breeding and natural selection. Pigeon breeders artificially select certain individual animals to reproduce. Nature, on the other hand, unconsciously selects individuals that are best suited to surviving in their local conditions. Given enough time, Darwin and Wallace argued, natural selection could produce new types of body parts, from wings to eyes.

After Darwin: A New Way to Study Life

Darwin's theory of evolution was enormous in its scope, and yet he remained vexed throughout his life by many biological mysteries. Heredity was an essential element of his theory, and yet he did not know how it was the offspring resembled their parents. The Czech monk Gregor Mendel was discovering genetics while Darwin was writing *The Origin of Species*, but Mendel's work languished in obscurity until the early 1900s. That rediscovery ignited an explosion of research into heredity, which eventually made it possible to track evolution on a molecular scale—a subject we'll return to in Chapter 5.

Geneticists were able to show that natural selection did indeed occur much as Darwin had proposed, but they did much more. As Darwin made clear in his writings, he recognized that selection was not the sole mechanism of evolution. And geneticists have confirmed that other mechanisms also drive evolutionary change. In a process called genetic drift, for example, genes and traits can become more common thanks simply to chance (Chapter 6).

In the remaining chapters of *The Tangled Bank*, we will encounter many scientists who are delving ever deeper into evolution, exposing the history of life and documenting its ongoing changes. Darwin played a profoundly important role in establishing the methods they use to study evolution, not just the concepts.

When Darwin came of age as a scientist, the concept of science was taking its modern form. (Indeed, the very word *scientist* did not rise in popularity until after about 1860.) Scientists use evidence to construct testable explanations about natural phenomena. They do not, in others words, simply pull away a curtain and reveal Truth.

For the most part, scientists are trying to make sense of things that remain hidden from view. When evolutionary biologists see a bee feeding on nectar from a flower—and picking up pollen to fertilize other flowers—they want

to better understand the millions of years of evolution that produced that partnership. They want to know why there are no 50-foot-long marine reptiles in the ocean today, even though those giants swam the seas for tens of millions of years. They want to turn back time.

Physicists likewise probe the invisible. They cannot see electrons with the naked eye, for example, and make personal observations of how they behave. They must run experiments to gather indirect clues about how electrons act and then use that evidence to come up with an explanation. Geologists cannot touch Earth's core. But they can eavesdrop on the reverberations of earthquakes as they bounce off the core and infer what it is made of. Epidemiologists track epidemics without ever seeing the vast majority of the viruses or bacteria that are making people sick. All scientists, in other words, seek to understand the invisible. Their mission is to find explanations that can account for indirect clues gathered through experiment and observation (FIGURE 2.17).



Michael Marsland/Yale University.

FIGURE 2.17

Scientists can study things beyond direct observation. Paul Turner of Yale University, for example, studies the evolution of viruses, which measure only about 100 nanometers across.

If all scientists have to go on are indirect clues, how can they ever know the explanations are correct? They must find a way to test the explanation against more evidence. The more accurate an explanation is in predicting the new evidence, the more confidence scientists have in it. If the prediction fails, scientists give their explanations a critical look and alter them to create a better one. In some cases, they can set up experiments to put those predictions to a test. If one scientist publishes an experiment that meets a

prediction, other scientists often try to come up with different experiments of their own to see if they get the same result.

This process is an essential part of science, because even an honest scientist can get a misleading result from an experiment. If an experiment is set up badly, it may produce a "false positive"—in other words, it meets a scientist's predictions, but not for the reasons the scientist thinks. On the other hand, experiments can also produce false negatives. A prediction may seem to fail, but only because the experiment wasn't set up carefully to test it. Later in this book, we'll consider a number of experiments evolutionary biologists have run to test predictions. Some scientists run their experiments in laboratories; others head out to lakes or forests to run them.

But scientists also study aspects of the world that cannot be subject to experiments. Astronomers study the formation of galaxies billions of years ago. Geologists reconstruct the birth of oceans and mountain ranges. It may not be possible to try to build a mountain range in a lab, but it is still possible to test predictions about things that occurred in the distant past. If paleontologists find the front half of a whale fossil, for example, they can predict that the rear half will also have traits found in whales and not—for example—have any traits that are unique to turtles. By studying differences in DNA, scientists can draw an evolutionary tree showing how species diverged millions of years ago. They can then test this hypothesis by adding a new species to the comparison or by analyzing a different set of genes to see if they get the same answer.

As we'll see in <u>Chapter 11</u>, the fossil record chronicles several mass extinctions, when most of the species in existence at the time disappeared in a few million years or less. No scientist lived through those mass extinctions long ago, but they can still develop hypotheses to explain them. Some 252 million years ago, for example, over 90 percent of all species disappeared (<u>page 289</u>). Some researchers have looked for clues to this so-called Permian-Triassic extinction in the chemistry of the rocks that formed at the time. They have proposed that the mass extinctions were caused, in part, by the disappearance of oxygen from the ocean, starting in deep water and spreading out to the coasts.

To test this hypothesis, paleontologists have looked for rocks from this period of time that reveal the timing and location of extinctions in the ocean. If the oxygen hypothesis were true, you'd predict that deep-water species

would disappear from the fossil record first, followed by shallow-water ones. Catherine Powers and David Bottjer at the University of California found just such a record. They studied fossils dating back to the Permian-Triassic extinctions. The fossils were formed by bryozoans, coral-like animals that anchor themselves to the seafloor and filter water. The scientists found that many deep-water bryozoan species went extinct first during the mass die-offs, and then shallow ones disappeared afterward—just as predicted (Powers and Bottjer 2007).

An Evolving Theory

This cycle of evidence, explanation, and testing can work effectively only on phenomena that follow reliable rules. Today, for example, the bonds that join atoms in a water molecule have the same energy as they did yesterday. You could imagine a world in which some supernatural force altered the bonds from moment to moment based on a mysterious whim. It would not be possible to use science to learn about how such a world works, because you would have no idea whether new evidence was produced by the same processes you were observing before.

This cycle gives rise from time to time to overarching explanations that account for a broad range of phenomena. These are theories. Many people think a theory is a hunch, a vague guess based on little evidence. When they hear scientists speak of "the theory of evolution," they assume that it's mere speculation, far less certain than a fact. But that's not what scientists mean when they speak of a theory. A theory is an overarching set of mechanisms or principles that explain a major aspect of the natural world. A theory makes sense of what would otherwise seem like an arbitrary, mysterious collection of data. And it is supported by independent lines of evidence that can be used to test each other.

Modern science is dominated by theories, from Newton's theory of gravity to the theory of plate tectonics to the germ theory of disease to Darwin's theory of evolution. Each of these theories came about when scientists surveyed research from experiments and observations and proposed an explanation that accounted for them in a consistent way. Scientists can use theories to generate hypotheses, which they can test with new observations and experiments. The more a scientific theory holds up to this sort of scrutiny, the more it becomes accepted. At the same time, however, many theories have changed with new evidence. Some parts of old theories are retained while taking on new dimensions as well.

Theories make sweeping claims. Molecular biologists in the 1950s came up with a theory that all organisms use a molecule called DNA to store genetic information. They based this theory on experiments on just a few

species. They didn't know for sure that all species encoded their genes in DNA. But given how consistently DNA turned up in those studies, scientists were reasonably confident that every animal, plant, fungi, protozoan, and bacterium also used DNA.

After five decades, scientists have found this to be the case in hundreds of thousands of species. But they have yet to test the theory on the millions of other species on Earth. Scientists have not tested every possible ramification of this theory, but that has not stopped them from accepting it as a good theory. Instead, they look to its predictive power. Every species scientists have looked at so far has DNA, and the theory that DNA is the storehouse of genetic information has allowed them to understand important things about how those species work.

The same rules apply to the theory of evolution. The modern theory of evolution still embraces many of Darwin's central insights, like natural selection and the tree of life. But it has matured and now takes a form that Darwin might not entirely recognize. That change is coming about as one generation of scientists after another examines the theory of evolution and tests aspects of it in specific ways, whether they are measuring natural selection in our species or running experiments to see how sexual selection influences mating success. Today the theory of evolution is as well supported as any other leading theories of modern science. But that does not mean that scientists know all there is to know about evolution. New fossils are discovered every year. The DNA of humans and other species is yielding profound secrets about how evolution works, and scientists are only beginning to understand them.

Still, the great expanses scientists have yet to explore do not diminish the importance of the theory of evolution. As with other theories, scientists value the theory of evolution for what it has helped them to understand so far. A good theory is like a powerful flashlight, helping scientists make their way into the dark. It's ironic that those who would reject evolution often call it a mere "theory," implying that it's inferior to facts. For scientists, just the opposite is true. A good theory is superior to a mere pile of facts, because it organizes them, changing them from a loose collection of details into a meaningful, well-supported picture.

Misconceptions about Evolution, Then and Now

No history of evolution would be complete without a look at the controversy it has attracted. When Lamarck and others began to champion evolution in the early 1800s, it immediately caused an uproar. When Charles Darwin first published *The Origin of Species*, scientists of the time engaged in fierce public debates over his theory. Scientists would not come to a consensus about some of the most important questions about the theory—such as how heredity makes natural selection possible—for more than 60 years after Darwin's book was published.

As these issues were settled, evolutionary biologists moved on to deeper questions and continued to debate each other. Today, when scientists meet at conferences to present their research on evolution, the disputes can get as fierce as they do at any scientific conference. Evolutionary biologists argue about the best way to reconstruct evolutionary trees. They argue about what caused mass extinctions. They argue about the relative importance of natural selection, genetic drift, and other processes in evolution. These debates can get spirited, and sometimes downright rough. But the biologists do not revisit long-settled subjects. They no longer debate whether life has evolved over billions of years, or that complex traits evolved in a stepwise fashion from earlier traits. It would be just as pointless for astronomers to revive debates about whether Earth revolves around the sun.

Yet opposition outside the scientific community has endured. In the early 1900s, an organized effort arose in the United States to remove evolution from classrooms. A century later, some people are still claiming that evolution is untrue. Some claim that the Earth is only 6000 years old, and that God created all life pretty much in its current form at that time. Others don't object to the notion that life is in fact billions of years old, but they claim that major features of biology are the result of something they call intelligent design—a planned creation by some kind of intelligent agent they claim not to be able to identify.

These claims are known collectively as creationism, because they all explain life's current diversity as the result of direct creation, rather than through natural processes such as natural selection. Evolutionary biologists reject creationism, because its objections to evolution have no basis in the evidence and because it is ultimately a nonscientific view of nature.

Many arguments for creationism begin with the construction of a strawman version of evolution. The straw man is knocked down, and its fall is supposed to be taken as evidence that evolution is false and creationism is true. Opponents of evolution often claim that it's false because evolutionary biologists have not discovered how life began. This is a case of apples and oranges. Evolution is a process that occurs when populations of organisms change genetically over time. Before life existed, there was no evolution. The origin of life is a vibrant field of research of its own—scientists are investigating what the oceans and atmosphere of the early Earth were like, and they are testing hypotheses for how prebiotic chemicals could have entered cycles of reactions that led to the earliest cells (Deamer 2011). But evolutionary biologists study what happened to life after it originated.

Sometimes opponents of evolution try to invoke physics to claim that it is simply impossible. Some claim, for example, that evolution violates the second law of thermodynamics, which holds that disorder increases in closed systems. In our everyday life, this law explains why we can never drive a perfectly efficient car and why a broken glass will never spontaneously reassemble itself. Yet the evolution of complex traits does not violate the second law of thermodynamics. That's because life does not evolve in a closed system. The sun provides an outside source of energy that allows life to grow, reproduce, and, ultimately, evolve (Styer 2008).

Creationists also challenge the evolution of complexity based on statistics. They claim that it's statistically impossible for evolution to have produced complex molecules like hemoglobin or organs like the eye. The odds of all the necessary mutations occurring are just too small. But this argument assumes that evolution is an entirely random process, which it is not. As we'll see in Chapter 6, natural selection spreads mutations through an entire population only if they have some beneficial effect. Many lines of evidence show that natural selection can, in fact, favor an entire series of mutations, which together give rise to new traits.

Some opponents of evolution argue that natural selection cannot produce innovations, because it simply favors some preexisting variants over others. It may favor a brown eye over a blue eye, for example, but it can't give rise to the eye itself. In fact, mutations do indeed provide the raw material for innovations. A gene can be accidentally duplicated, for example, or it may switch to respond to a different signal, producing a new circuit of genes that can take on a new function. Old adaptations selected for one function can switch to a new one.

Another straw-man argument is the supposed absence of fossils that document major transitions in evolution. Because biologists can't point to fossils that document every single intermediate stage in evolution, they supposedly cannot claim that new species evolved from old ones. This criticism ignores the process by which fossils form, which we'll look at more closely in the next chapter. It's inevitable that the fossil record is incomplete, and yet, thanks to the hard work of thousands of paleontologists, we keep learning more about evolutionary transitions—such as the origin of whales, which was fairly mysterious before 1980.

Understanding evolution helps us make sense of the mechanisms underlying the patterns of fossils and living species, and it guides us toward new evidence. Creationism in its various forms, on the other hand, lacks this sort of explanatory power. Things are the way they are simply because they are. What explains the presence of the 13 species of finches with different beaks on the Galápagos? They were created that way.

To say that species were created directly by God, as many creationists do, is to make a religious claim, not a scientific one. Scientists cannot evaluate a theory about a supernatural agent, since scientific theories explain phenomena that follow natural, repeatable patterns. To invoke an intelligent designer rather than a divine creator does not help. The designer is either supernatural or natural. If the designer is supernatural, the claim of intelligent design is not scientific. If the designer is natural, it should be possible to make predictions about how it has produced life's diversity. But since we can't know the nature of the designer, we're told, this claim reaches a scientific dead end.

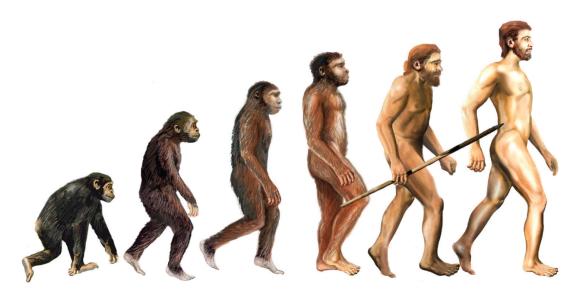
Creationists do not have a monopoly on misconceptions about evolution. In his own day, Darwin watched with dismay as some people embraced his books, but then came to unwarranted conclusions. For example, in the late

nineteenth century there arose a movement, sometimes called social Darwinism, which argued that those who succeeded in business were more "fit" than the poor, and their success was good for the entire species. Though Darwin was a man of his time—with many of the prejudices of an upper-class Victorian Englishman—he was opposed to using his scientific research to justify poverty or slavery (<u>Browne 2006</u>).

Darwin also looked askance at those who would celebrate humans as the pinnacle of evolution. In fact, he rejected any kind of hierarchy in life. "It is absurd to talk of one animal being higher than another," he once wrote. "We consider those, where the intellectual faculties most developed as highest," he remarked, and yet he imagined that a bee might disagree, holding instinct to be more important. Darwin recognized that every living species is the product of evolution, which has adapted it to its environment. Even today, however, it's still common to hear people describe bacteria or fungi as a "lower" form of life, even though these organisms have evolved into vastly more species than animals have and can live in a far wider range of environments.

The notion of higher and lower forms of life has very deep roots. Medieval European theologians envisioned a "Great Chain of Being," with plants at the base and humans at the top—and angels above them. In a sense, Lamarck turned the Great Chain of Being into the Great Elevator of Being, envisioning the history as a never-ending progress from the simple to the complex. Even today, it's not uncommon to hear evolution described in such terms, as if it always proceeded from simple to complex. Complex traits have indeed evolved from simpler precursors. In Chapter 10, for example, we'll look at evidence for the evolution of our eyes from light-sensitive cells. But that example doesn't mean that all evolution is simply a progression toward the complex. After all, bacteria evolved at least 3.5 billion years ago, and most of those lineages did not turn into plants or animals. What's more, evolution can make complex things simpler. Our ancestors, for example, lost their tails and have only vestigial bones left at the base of the spine. Many species of free-living bacteria have evolved to become full-time residents of host cells, and in the process they've jettisoned many of the genes they once needed to make complex molecules of their own. Now they can just grab those molecules from their hosts.

A variation on this march of progress is also one of the most common images of evolution, shown in FIGURE 2.18. This image encourages us to think of evolution as a linear process, moving from one ancestral state to one evolved state. Darwin had a much more accurate conception of evolution when he drew a tree in his sketchbook. Lineages branch apart, and evolution can work differently in each one. When our ancestors branched off from the ancestors of chimpanzees some 6 million years ago, they did not evolve in a single lineage to our current form. As we'll see in Chapter 14, our lineage gave rise to perhaps 20 species of bipedal apes, each with its own particular set of adaptations, all of which have become extinct—with the sole exception of our own.



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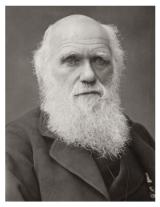
FIGURE 2.18

This common image of evolution promotes a misleading view of the process. Evolution is not a steady march of progress toward some predetermined goal. A better image of the evolutionary process is a tree sprouting branches, as Darwin sketched in his notebook, as shown in <u>Figure 2.13</u>.

The image of the march of progress can lead people to misunderstand how scientists actually study evolution. If an important new fossil comes to light, for example, headlines will often describe it as a "missing link." In fact,

paleontologists generally don't study evolution by looking for direct lines of ancestors and descendants. They infer the evolutionary relationships between species by comparing their anatomy or their DNA. The history of life does not unfold in a line: it is encoded in billions of branches of evolution.

In the twenty-first century, evolutionary biology has become a remarkably rich body of knowledge, extending well beyond *The Origin of Species*. But biologists do not think less of Darwin as a result. After all, we think no less of Isaac Newton for not knowing about the hidden structure of atoms. Just as Newton opened a door for generations of physicists, Darwin opened a door for biologists.



Huntington Library/ Superstock, Inc.

FIGURE 2.19

Charles Darwin died in 1882. Since his death, biologists have made many discoveries about life that have given them a better view of evolution.

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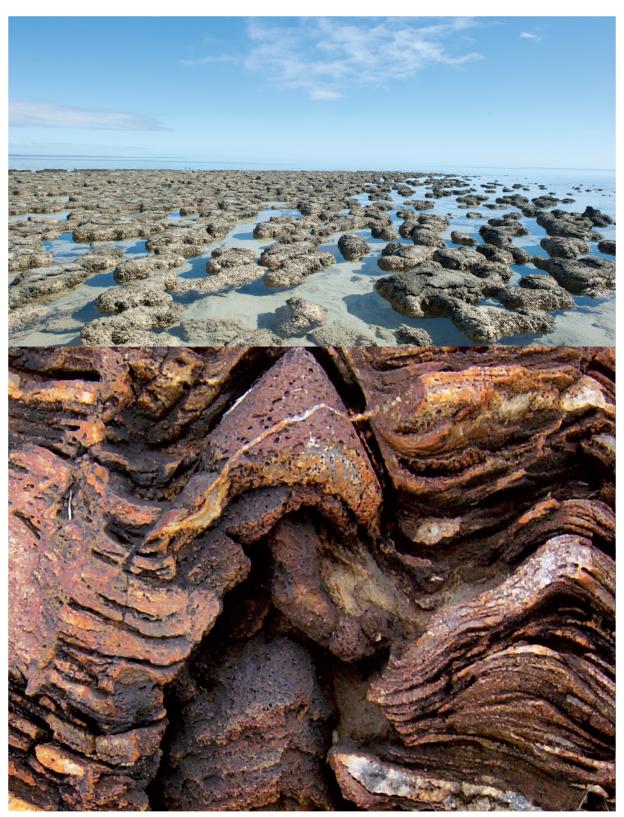
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How Geology and Paleontology Reveal the History of Life



Top to bottom: Rob Bayer/Shutterstock; Abigail Allwood.

Top: Stromatolites are layered mats of bacteria. They are relatively rare today. (The ones shown here live on the coast of Australia.) Bottom: Stromatolite fossils in Australia dating to over 3.4 billion years ago are among the earliest evidence of life on Earth.

Abigail Allwood's job sounds like a contradiction in terms: she's a paleontologist who works at NASA's Jet Propulsion Laboratory. But there's actually a sound reason that NASA keeps paleontologists like her on staff. When NASA explores other planets, they want to know how to recognize any fossils left behind by extraterrestrial life. Allwood does not look for just any fossil, however. She goes to one of the most remote, inhospitable places on Earth: deep into the Outback of Australia. Plenty of lizards and cockatoos live there, but virtually no people. Water is scarce among the bare outcrops and hills, and the days can be scalding hot. The name of the geological formation where Allwood works is a grim joke: North Pole.

Allwood hikes along the exposed rocks, taking photographs and sometimes hammering off pieces to take home to study further. There's nothing in the rocks that looks alive to the inexperienced eye. The most notable thing about them is that they are made up of fine, even layers, which curve and sag into strange shapes. Some look like upside-down ice cream cones, and others look like egg cartons.



Abigail Allwood.

FIGURE 3.1

Geologist Abigail Allwood studies fossil stromatolites to get clues about the earliest period of life on Earth.

As inscrutable as these rocks may seem, Allwood's research indicates that they were formed by living things. The North Pole once was a broad, shallow sea where lush mats of bacteria, called stromatolites, stretched for miles. The particularly striking thing about the fossils is their age. They are 3.43 billion years old, an age that makes them some of the oldest traces of life on Earth.

Allwood is one of thousands of scientists who traverse the planet in search of traces of the history of life. Together, they are creating a record—from fossils, molecules, even atoms—that chronicles how life first emerged on Earth, how it flourished, diversified, suffered extinctions, and continued to change for at least 3.5 billion years, giving rise only 200,000 years ago to a new species of upright apes: our own species, *Homo sapiens*.

The Ancient Earth

Charles Darwin is now best known as an evolutionary biologist, but he first came to fame as a geologist. On his journey aboard the HMS *Beagle*, he made careful observations of mountains, islands, and other geological formations. He recognized that these formations were the result of gradual changes spread over vast amounts of time. Like many other nineteenth-century geologists, Darwin rejected the widely accepted belief that the world was only a few thousand years old.

Darwin and his fellow geologists could not determine exactly how old a particular fossil or rock might be, however. They could make only rough estimates of the time it had taken for a geological formation to emerge, by observing how quickly sediments accumulated on riverbanks and coastal waters. While working on *The Origin of Species*, Darwin applied this method to the Weald, a stretch of ridges and valleys in southeastern England. He concluded that the area was formed through gradual erosion over the course of 300 million years. If it had taken hundreds of millions of years for a relatively small geological formation to reach its current state, Darwin surmised, then the Earth itself must be billions of years old.

Darwin drew sharp criticisms from some quarters for these geological claims. The most famous of his critics was the eminent physicist William Thomson (Lord Kelvin). Kelvin argued that the world could not be as old as many geologists proposed. His argument was based not on formations of rocks, but on their temperature.

Let's assume that the Earth began as a ball of molten rock, Kelvin said. The crust would rapidly cool and harden; then the interior heat would flow through it to escape into space. Since a hot rock cools at a steady rate, Kelvin reasoned that you could use the current temperature of rocks to estimate how long they had been cooling. Rocks on the planet's surface would not give a reliable estimate, because they were heated by the sun every day and cooled every night. But the rocks at the bottom of mine shafts stayed at the same warm temperature year-round. Kelvin obtained readings from those rocks

and then used them to estimate how long the Earth had been cooling. He concluded that at most, Earth could be only 20 million years old.

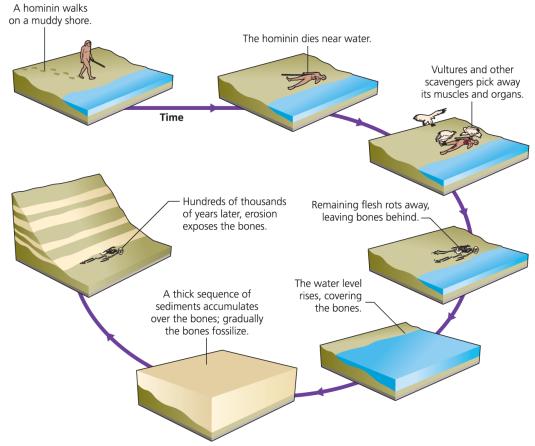
Kelvin, as it later turned out, was wrong. To calculate Earth's heat flow, he had assumed the planet was a rigid sphere. In the twentieth century, geophysicists would discover that the planet's interior is dynamic. Hot rock rises through the mantle, cools, and then sinks back down again. This movement drives the motion of tectonic plates across the surface of the Earth. It also makes the upper layers of the Earth warmer than proposed in Kelvin's model (England et al. 2007).

In the early 1900s, physicists finally found a way to measure the absolute age of rocks. They discovered that radioactive atoms decay into other elements at a precise rate. By measuring the products of this decay in rocks, scientists can estimate how long ago the rocks formed (see **BOX 3.1**). Radiometric dating has revealed that Earth formed 4.567 billion years ago as part of a dust cloud around the sun (<u>Wood 2011</u>). This dating technique also allows scientists to estimate the ages of rocks in which fossils are found. The fossil record now stretches back at least 3.5 billion years. Darwin, in short, turned out to be right, and Kelvin was wrong.

A Vast Museum

Darwin recognized that evolution made sense of the fossil record. But he also knew that some critics would try to use the fossil record to challenge him. Why, they might ask, hadn't paleontologists found fossils from every stage of evolution from one species to another? "I believe the answer mainly lies in the record being incomparably less perfect than is generally supposed," Darwin wrote in *The Origin of Species*. "The crust of the Earth is a vast museum; but the natural collections have been imperfectly made, and only at long intervals of time."

Over the past 150 years, scientists have learned a great deal about how organisms fossilize—and, more often, fail to fossilize. To understand this process, they've observed how dead organisms decay over time, and they've replicated some of the chemistry that turns living tissues into rock. FIGURE 3.2 illustrates a typical sequence of events. An organism dies and is scavenged by carrion feeders. Microbes infest the cadaver, and eventually all that's left is the skeleton. A portion of the skeleton is buried in sediments and ash. Water percolating through the sediment pores delivers minerals that gradually fill the latticework of a bone or a shell. Over thousands of years, the sediment turns to rock, and the organism becomes a fossil.



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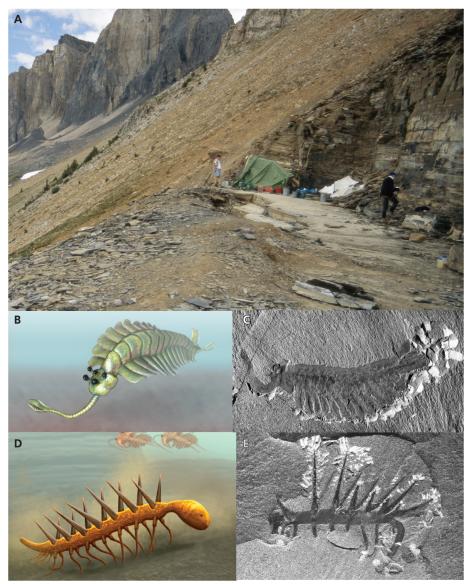
FIGURE 3.2

After organisms die, their bodies are sometimes covered by sediment and then gradually turned to minerals, leaving behind fossils. (Information from <u>Prothero 2007</u>.)

There are plenty of opportunities along the way for this process to fail. A skeleton may be so badly trampled, sun-beaten, and rain-soaked that nothing is left to become a fossil. Even after a fossil forms, pressure and heat may destroy it. Erosion can remove overlying rock and expose the fossil again; wind and rain can then gradually blast it away. The only chance for paleontologists to rescue this fossil from oblivion is the narrow window of time between its exposure and its destruction. It's thus no surprise that the fossil record is far from complete.

In certain very rare situations, soft tissue such as muscles or hair can also become fossilized. In even rarer situations, an entire community of soft-bodied organisms may be preserved. In 1909, for example, Charles Doolittle

Walcott discovered an extraordinary lode of fossils in the mountain slopes of British Columbia. Quarries at this site, dubbed the Burgess Shale, have yielded more than 65,000 specimens of marine animals—mostly soft-bodied—representing at least 93 species, all of which died 505 million years ago. The animals were preserved so well thanks to where they had lived: a reef on a steep mudbank. Sometimes the bank collapsed, and the mudslides would hurl the animals into the abyss, where the water was nearly free of oxygen. In those cold, oxygen-free depths, bacteria were unable to decompose the animals, and so their soft tissues were preserved in exquisite detail (Briggs, Erwin, and Collier 1995). The Burgess Shale has thus become one of the most important places on Earth to learn about the early evolution of animals (FIGURE 3.3).



A: L. Newman & A. Flowers/Science/ardea.com; B: Quade Paul; C: The Smithsonian Institution/Chip Clark; D: Carl Buell; E. The Smithsonian Institution/Chip Clark.

FIGURE 3.3

A: A fossil site in the Canadian Rockies called the Burgess Shale has yielded vast numbers of fossil animals dating back 505 million years. B–C: Among the animals preserved in the Burgess Shale is *Opabinia*. D–E: Another bizarre animal is *Hallucigenia*.

Bringing Fossils to Life

Once paleontologists find a fossil, they bring it to their lab and examine it for clues about its existence. They can inspect its anatomy for traits they can use to place it on the tree of life (<u>Chapter 4</u>). And they can make inferences about how the fossil lived—how it walked or flew, how it found food, how it reproduced and reared its young (<u>FIGURE 3.4</u>).



A. Senckenberg, Messel Research Department, Frankfurt a. M. (Germany); B. The Trustees of the Natural History Museum, London; C. age fotostock/age fotostock.

FIGURE 3.4

Fossils can preserve clues about the behavior of extinct animals. A: Two turtles dating back about 47 million years died while mating in a poisonous lake. B: A marine reptile called an ichthyosaur gives birth to a live offspring, rather than an egg. C: A fish fossilized in the midst of eating another fish.

Radioactive Clocks

When physicists discovered the structure of atoms in the early 1900s, they also made it possible for geologists to precisely measure the ages of rocks.

All atoms are made of three fundamental particles: protons, neutrons, and electrons. The number of protons in an atom determines which element it belongs to, but the number of neutrons can vary. For example, all carbon atoms have six protons. While 98.93% of all carbon atoms on Earth have six neutrons, 1.07% have seven, and one carbon atom in every trillion has eight. These so-called isotopes of carbon are named, respectively, carbon-12, carbon-13, and carbon-14.

In some arrangements, the protons and neutrons in an atom are perfectly stable, but in other arrangements they will sooner or later fall apart. Along the way, the atoms change from one element to another. Uranium-238 breaks down by releasing a pair of neutrons and a pair of protons, thereby turning into thorium-234. Thorium-234 decays in turn to protactinium-234. Uranium-238 passes through 13 of these intermediate states before finally reaching a stable form: lead-206.

Each radioactive isotope has a distinctive decay rate, known as its half-life. The half-life of uranium-238 is 4.47 billion years, which means that in this period of time, half of a given quantity of uranium-238 will decay into lead-206. It also means that in each year, any given atom of uranium has a very tiny chance of decaying -1.55125×10^{-10} to be exact (Lanphere 2001).

Earth's rocks contain small amounts of radioactive material. When fresh lava cools and forms new igneous rocks, trace amounts of radioactive atoms get locked into their minerals. Imprisoned in the rocks, the atoms continue to decay. The older the rock gets, the fewer original radioactive atoms remain (Dalrymple 1991). Geologists have developed a number of methods for telling time with radioactive isotopes.

In one common technique, geochronologists measure the age of a rock by taking advantage of rubidium-87, which decays into strontium-87 with a half-life of 48.8 billion years. You can't determine the age simply by measuring these two isotopes. To see why, imagine you found a rock that contains equal amounts of both isotopes. You might conclude that half of the rubidium-87 present when the rock formed has since decayed into strontium-87. That would mean the rock is 48 billion years old—over three times older than the universe itself.

This absurd figure is the result of a mistaken assumption—namely, the assumption that all the strontium-87 we measure in the rock must be the product of radioactive decay of the rock's own rubidium-87. In fact, when a rock forms, it may incorporate strontium-87 isotopes from the start. The decay of the rock's rubidium-87 may add only a tiny extra amount of strontium-87 to its original supply.

If we knew how much strontium-87 the rock started out with, estimating the age of the rock would be easy. We'd be able to subtract the original portion of strontium-87 present in the rock to calculate how much was added by radioactive decay. Then we could use the half-life of rubidium-87 to determine the age of the rock. Unfortunately, no one was around when the rock formed to measure its strontium-87, so this method is impossible. Fortunately, the rock itself gives us a way to get around this problem.

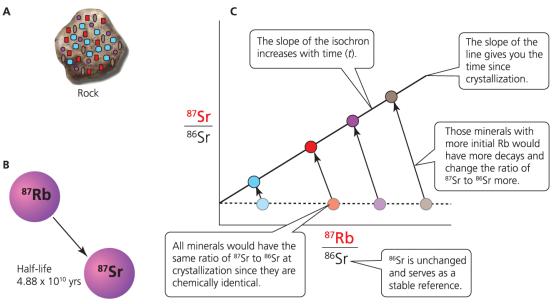
The secret to this trick is that the rock's strontium comes in two isotopes. The strontium-87 in a rock is the result of radioactive decay, either before or after the rock formed. But the rock will also contain strontium-86, which is not a product of decay. The two isotopes of strontium behave identically in chemical reactions. The only difference is that the supply of strontium-86 will stay the same in the rock, while strontium-87 will creep upward thanks to the decay of rubidium-87.

To measure the age of the rock, we can measure both strontium isotopes in many different parts of the rock. Some of the parts may have only a little strontium while other parts are rich in the element. No matter how much strontium any particular part has, however, it will have the same ratio of strontium-87 to strontium-86 when the rock forms. As the rock gets older and rubidium-87 becomes strontium-87, the ratio increases in every part of the rock.

We still aren't ready to estimate the age of the rock. We need one more piece of information: the amount of rubidium-87 in the different parts of the rock. Since rubidium and strontium are different elements, they behave differently in chemical reactions. This means that when a rock forms, different parts will form with different ratios of rubidium and strontium. But in every part, the rubidium-87 steadily decays. As a result, the ratio of rubidium-87 to strontium-87 steadily drops.

We can visualize these changes on a graph, as illustrated in **FIGURE 1**. We plot each part of the rock we analyze: the ratio of rubidium-87 to strontium-86 is on the x-axis, and the ratio of strontium-87 to strontium-86 is on the y-axis. Since the strontium isotopes are chemically identical, their ratio starts out the same throughout the rock. As a result, we end up with a straight line, which geochronologists call an isochron. Over time, the strontium-87 to strontium-86 ratio increases thanks to radioactive decay, so each point on

the graph climbs up the y-axis. Meanwhile, the rubidium-87 supply is falling, so each point is also moving to smaller values on the x-axis.



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FIGURE 1 A: A rock contains minerals that have different amounts of rubidium and strontium. B: Rubidium-87 decays to strontium-87. After a rock crystallizes, this radioactive decay increases its supply of strontium-87. C: When a rock first forms, the ratio of strontium-86 to strontium-87 is the same throughout the rock, regardless of the proportion of rubidium to strontium. The values form a straight horizontal line, called an isochron. As the strontium-87 decays, the ratios change, increasing the slope of the isochron. (Information from <u>Lanphere 2001</u>.)

How far each part of the rock moves on the graph depends on how much rubidium-87 it starts out with. A part of the rock with little rubidium-87 will not gain much more strontium-87. In a part of the rock that starts out with a high ratio of rubidium-87 to strontium-86, the changes will be larger.

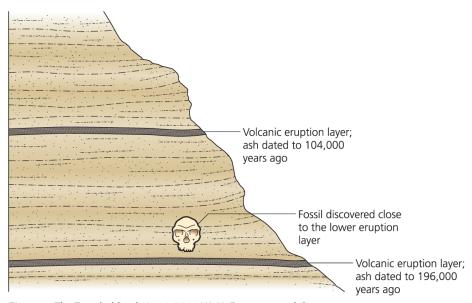
Together, these changes produce a remarkably simple change: the isochron turns in a counterclockwise direction. The older the rock gets, the further the isochron turns. By measuring the steepness of the isochron, we can estimate the age of the rock.

Geochronologists can measure different time scales by measuring different elements. Rubidium-87's long half-life makes it good for measuring very old rocks. Potassium-40 takes only 1.25 billion years to break down into argon-38, and so it provides a more

accurate clock for dating younger rocks. In some cases, scientists can measure two different isotopes in the same rock, and they typically end up with the same estimate from both methods. The fact that they can derive the same age with independent methods confirms that radiometric dating works.

By dating rocks, scientists can estimate the ages of fossils they contain. If a fossil is sandwiched between layers of volcanic ash rich in potassium-40, for example, the layers can create upper and lower bounds for the age of the fossil itself. In 1967, scientists discovered fossils of humans at a site near the village of Omo, Ethiopia. The scientists knew the fossils were old, but it was hard to determine just how old they were. Almost three decades later, a team of scientists went back to the site to take a closer look. They discovered two layers of volcanic ash, one above the rocks where the fossils had been found and another right below them.

The argon in the upper layer yielded an age of 104,000 years, with a margin of error of 7000 years. The lower layer was 196,000 years old, with a margin of error of 2000 years. A careful study of the sediments between those two layers indicated that the fossils were closer to the older boundary than to the younger one—perhaps as old as 195,000 years. Thanks to this research, the Omo fossils became the oldest known fossils of members of our own species (McDougall et al. 2005).



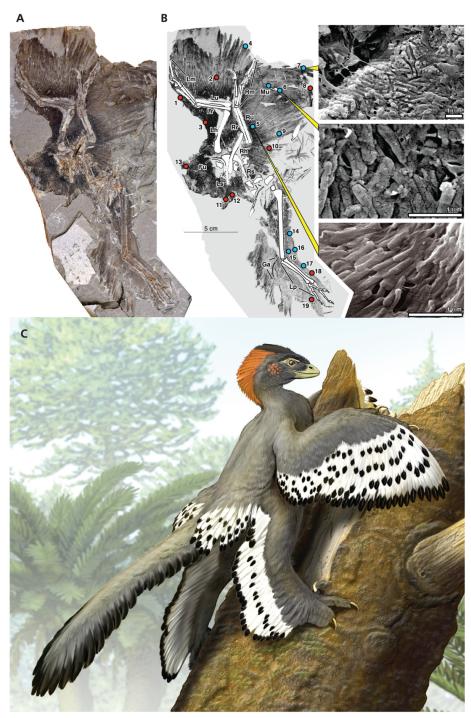
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FIGURE 2 Paleontologists use as many lines of evidence as possible to estimate the age of fossils. By calculating the ages of layers of volcanic ash above and below a fossil, they can establish upper and lower bounds for when it formed. In Ethiopia, the oldest

fossils of *Homo sapiens* are sandwiched between two layers of volcanic ash. Using potassium-argon dating to estimate the age of the layers, the scientists determined that the fossils were about 195,000 years old.

Jakob Vinther, a paleontologist now at the University of Bristol, and his colleagues have recently used fossils to figure out the colors of ancient feathers. In living birds, some of the colors in their plumage are produced by microscopic structures called melanosomes. The melanosomes are packed with pigment, and their shape and arrangement determine how light reflects off the pigment. Vinther and his colleagues discovered that fossil birds have exactly the same melanosomes in their feathers—and thus can infer their color as well (Vinther et al. 2008).

As we'll see in the next chapter, birds evolved from ground-running dinosaurs. Over the past 20 years, paleontologists have found many fossils of these dinosaurs with feathers preserved on their bodies. Vinther and his colleagues have put some of these dinosaur fossils under their microscopes, and they've found melanosomes in them as well. Based on the pattern of those melanosomes, the scientists have concluded that dinosaurs had bold plumage, as shown in FIGURE 3.5 (Vinther et al. 2010; but see also McNamara et al 2013).



A. Jakob Vinther; B. 2010 American Association for the Advancement of Science; C. Carl Buell.

FIGURE 3.5

A: Fossils of a 150-million-year-old dinosaur called *Anchiornis huxleyi* include preserved feathers. B: The feathers include cellular structures called melanosomes that help produce color. Jakob V inther and his colleagues took small samples from an *Anchiornis*

fossil marked by the dots shown here. The size, shape, and organization of the melanosomes allowed them to reconstruct the original color of the feathers. C: *Anchiornis* had a striking plumage, which may have been important for courtship displays.

Traces of Vanished Life

The fossil record is made up of much more than just fossil bones and shells. About 300 million years ago, for example, giant swamps spread across many of the continents. When plants died there, they did not immediately decay. Instead, they fell into the swamps and were rapidly buried in sediment. Bacteria then began to break them down. Eventually, the swamps were drowned by rising oceans and then buried under vast amounts of marine sediment. The plant material was transformed yet again, under tremendous pressure and heat, until it took on the familiar black, hard form we know as coal. Scientists can get clues to the plants that gave rise to the coal by examining it closely. Sometimes they even find stems and other intact pieces of plants in it.

In certain rocks, it's even possible to identify individual molecules from organisms that lived billions of years ago; these molecules are known as biomarkers (<u>Gaines 2008</u>). To identify a true biomarker, scientists must go to great lengths to show that a molecule in a rock couldn't have formed through a biology-free process. Our bodies make amino acids, for example, but that doesn't mean that amino acids found in a rock were produced by an ancient organism. Many chemical reactions on Earth can give rise to amino acids. Astronomers have detected amino acids even in interstellar clouds.

Other molecules show clear signs of a biological origin. Our bodies can assemble hundreds of amino acids into a single protein, for example, using an army of enzymes to carry out the complex chemistry. In the absence of biology, there's no evidence that such proteins ever form on their own. When scientists find biomarkers in a rock, they can sometimes even infer what kind of organism produced it. Okenane, for example, is a pigment produced by purple sulfur bacteria, a group of species called Chromatiaceae (FIGURE 3.6). Chemists know of no reactions that take place on Earth in the absence of the bacteria that can produce this molecule. Jochen Brocks, of Australian National University, and his colleagues have found okenane in 1.64-billion-year-old rocks in Australia.

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Paleontologists have found a molecule called okenane in Australian rocks. The only natural way it can be produced is by purple sulfur bacteria. The bacteria produce a red pigment called okenone, which later undergoes reactions that turn it into okenane. Thus okenane is a biomarker, and it indicates that purple sulfur bacteria had already evolved when the rocks formed 1.64 billion years ago.

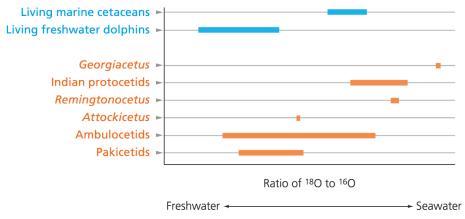
Research by Brocks suggests that purple sulfur bacteria existed at 1.64 billion years ago. That's a fascinating idea, because purple sulfur bacteria are rare today, found only in extreme environments with low levels of oxygen and high levels of sulfur. Their abundance 1.64 billion years ago supports the hypothesis that the oceans at the time were toxic—to organisms like ourselves, at least (Brocks and Banfield 2009).

Even the individual atoms in rocks can offer scientists clues about ancient life. All atoms belonging to the same element have the same number of protons. All carbon atoms have six protons, for example. Most carbon atoms have six neutrons, but a small fraction of them have seven. These isotopes, as they're called, are handled differently by living organisms. Plants, for example, obtain their carbon from the atmosphere, incorporating a mixture of carbon-12 and carbon-13 isotopes into their biomass. Because carbon-13 is heavier than carbon-12, it's more difficult for the plants to absorb it. As a

result, the ratio of carbon-13 to carbon-12 is lower in plants than it is in the atmosphere.

Different plants have slightly different ratios of carbon isotopes, depending on how they carry out the process of photosynthesis. Most plant species carry out C_3 photosynthesis, so named because it incorporates carbon dioxide into a molecule with three carbon atoms. Grasses and certain other plants carry out C_4 photosynthesis, in which the carbon dioxide is incorporated into four-carbon molecules. Thanks to the differences in their biology, C_4 plants have lower levels of carbon-13 than C_3 plants. When scientists measure the ratio of carbon isotopes in plant fossils, they find the same difference between extinct C_3 and C_4 plants. A cow that feeds on grasses, which are C_4 plants, will incorporate their carbon atoms into its own cells. As a result, the fossil of a cow will end up with a low level of carbon-13, reflecting its diet.

Hans Thewissen, the whale expert we met in <u>Chapter 1</u>, has used isotopes to track the transition of whales from land to water. Instead of carbon, he examines oxygen. While most oxygen atoms have eight neutrons, a small fraction of them have 10, and seawater has a higher ratio of oxygen-10 than freshwater. Animals that live on land incorporate oxygen from freshwater into their bones, while marine animals use seawater oxygen. As a result, marine animals have "heavier" oxygen in their fossils than terrestrial ones do. Living whales and dolphins have a larger percentage of heavy oxygen in their bones than do mammals that live on land (<u>FIGURE 3.7</u>).



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Paleontologists can get clues about extinct organisms by examining the isotopes in their fossils. Whales and dolphins incorporate into their teeth the oxygen atoms from the water they live in. Sea-water has more oxygen-18 than freshwater does, and this difference is reflected in the teeth of living whales and dolphins that live in each habitat. Hans Thewissen and his colleagues have measured the isotopes of fossil whales to infer where they lived. As this graph shows, the earliest whales were adapted to living in or around freshwater. *Ambulocetus*, which had a more amphibious anatomy, had an oxygen ratio that spanned the two environments. They may have lived in brackish waters or traveled between the open ocean and rivers. Later whales had a ratio you'd expect from seawater, suggesting they had adapted completely to the ocean. (Information from Thewissen and Bajpai 2001.)

Thewissen wondered if the oxygen atoms in ancient whale fossils might indicate where they lived. So he and his colleagues ground up tiny samples of ancient whale teeth and measured the ratio of light and heavy oxygen. They discovered that *Pakicetus* still drank freshwater. *Ambulocetus*, which belongs to a younger branch of the whale tree, had an intermediate ratio, suggesting that it was drinking brackish water near the shore, or a mix of freshwater and sea-water. More recent fossil whales had the ratios you would expect in animals that drank seawater alone. Together, these isotope ratios chart a transition from land to estuaries to the open ocean—the same transition documented in the changing shape of their skeletons (Thewissen 2001).

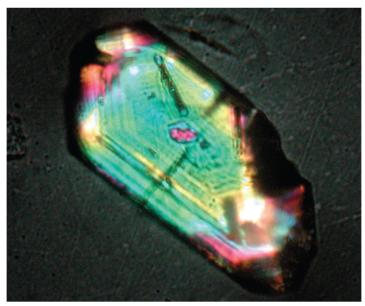
Life's Earliest Marks

We can use all these lines of evidence—fossils, biomarkers, isotopes, and so on—to reconstruct a chronology of life on Earth. Obviously, any version of this chronology is just a tentative version that will have to be revised as new evidence comes in. Indeed, some of the most important discoveries scientists have made about the history of life have been made in just the past decade.

When the sun first formed, it was surrounded by a primordial disk of dust and other materials. Gravity caused the dust to clump together into small bodies, called planetesimals, that then collided to form planets. When our planet formed 4.567 billion years ago, it was molten. Over millions of years it slowly cooled, its molten crust hardening and lighter rock formations rising to become the first continents. Gases escaped from the rocks to form Earth's atmosphere. Water arrived on the surface of the planet, most likely delivered by comets and asteroids. The basins between the continents filled with the water, forming oceans.

For hundreds of millions of years, Earth collided with debris remaining from the original solar disk. One such collision was so big that the rocky rubble thrown up from the impact began to orbit the Earth and eventually coalesced to form the Moon. The giant impacts began to taper away about 3.8 billion years ago. Over the next billion years or so, the crust of the planet broke into plates. Hot rock rose up in some of the cracks between the plates and added to their margins. Meanwhile, the opposite margins of the plates were driven down under the crust, heating up as the plate descended until it mixed into the underlying rock.

This combination of bombardment and burial has swept away almost all of Earth's original surface (Sleep 2010). The only traces of the crust's first few hundred million years are preserved in microscopic crystals known as zircons (FIGURE 3.8). Zircons can't preserve fossils, but they can preserve isotopic clues to the chemistry of the early Earth.



Stanford University/Getty Images.

Tiny specks of carbon can be preserved for billions of years in minerals known as zircons. The balance of carbon isotopes can provide clues to what life was like when they were trapped in the mineral.

Before life began, the only source of carbon on the surface of Earth would have come from lifeless sources, like volcanoes. But once life emerged on Earth, it would have produced abundant amounts of organic carbon, which gradually would have become incorporated into sedimentary rocks. Scientists can distinguish between the two sources of carbon, due to biology's preference for lighter carbon isotopes.

In 2004, Minik Rosing and Robert Frei of the University of Copenhagen announced they had found geological evidence of this shift from non-biological carbon to biological (<u>Rosing and Frei 2004</u>). They extracted bits of 3.7-billion-year-old carbon from rocks in Greenland and discovered a ratio of carbon isotopes that suggested it had come from a living source. Rosing and Frei concluded that these rocks hold the earliest sign of life, produced most likely by photosynthetic bacteria.

Some researchers have challenged Rosing and Frei's interpretation. They've argued that geological processes could have created the ratio of carbon isotopes in the rocks—without any need for life (Westall 2008). Such

uncertainty hovers over much of the earliest evidence for life on Earth. In the 1980s in Australia, J. William Schopf of UCLA discovered what he proposed were 3.5-billion-year-old fossils of bacteria. Martin Brasier of the University of Oxford has argued that Schopf's fossils were actually formed by tiny blobs of mineral-rich fluids (Brasier et al. 2006).

To better understand the early history of life, scientists are continuing to scour ancient rocks. Abigail Allwood and her colleagues discovered their strange, egg-carton-like rocks in some of the oldest geological formations on Earth. The researchers then found striking microscopic similarities between the rocks and large mounds built today by colonies of bacteria. These structures are known as stromatolites. Today, stromatolites survive in hot, salty, shallow lagoons. In any other habitat, they'd be devoured by animals and protozoans. Despite their rarity today, stromatolite fossils are abundant in the early fossil record.

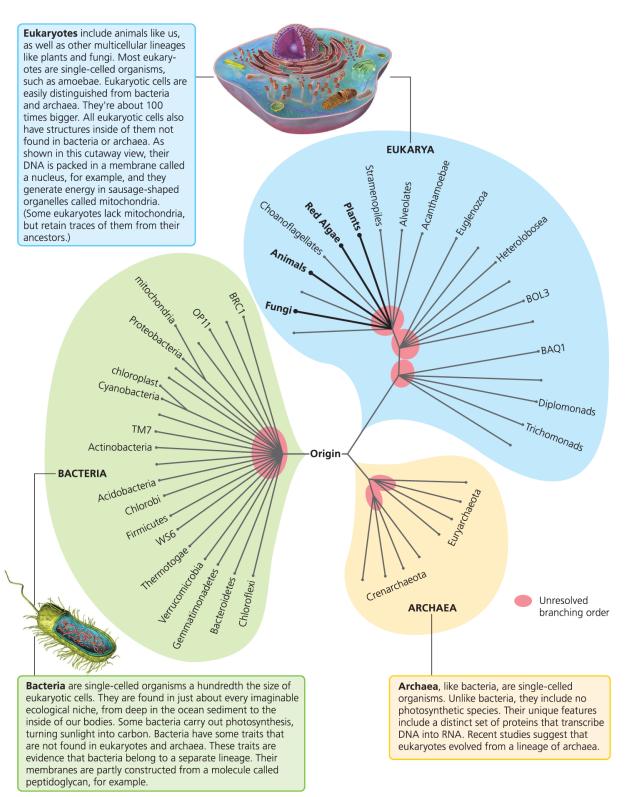
In 2006, Allwood and her colleagues published a description of the rocks they had found and declared them to be the remains of 3.45-billion-year-old stromatolites. If they're right (and many of their colleagues think they are), they may have found evidence of some of the earliest life on Earth (Allwood et al. 2006; Bontognali et al. 2012; see also Wacey 2010).

The Rise of Life

In *The Origin of Species*, Charles Darwin observed that the oldest fossils known in the mid-1800s belonged to animals that lived during the Early Cambrian period, which we now know lasted from 542 to 510 million years ago. If Darwin's theory was right, then life must have been evolving long beforehand. "During these vast periods the world swarmed with living creatures," he wrote.

Yet Darwin recognized that no fossils of those creatures had yet been found. "To the question why we do not find rich fossiliferous deposits belonging to these assumed earliest periods prior to the Cambrian system, I can give no satisfactory answer," he wrote. Today we know the answer Darwin could not provide: the fossils had yet to be discovered. Scientists have found fossils dating back almost 3 billion years earlier than the Cambrian period, and they are piecing together a fossil record that becomes richer each year (Knoll 2003).

During those first 3 billion years, life remained mostly microscopic. Yet paleontologists can recognize the first appearances of some of the major lineages of life. Molecular biologists have drawn a tree of all life, based on the DNA of living species. As shown in FIGURE 3.9, the tree is made up of three main groups: Eukarya, Bacteria, and Archaea. (See Chapters 4 and 9 for more details on building trees from DNA.)



Zimmer, The Tangled Bank, 2e, © 2014 W. H. Freeman and Company

This evolutionary tree shows how the major lineages of life are related to each other. Most biologists argue for three major divisions to the tree: Bacteria, Archaea, and Eukarya. The dates on some of the nodes on the tree show the earliest fossil evidence for each lineage. (Information from Pace 2009.)

Eukaryotes include multicellular lineages such as animals, plants, and fungi. But they also include a wide range of single-celled lineages known as pro-tists. Eukaryotic cells are easily distinguished from both bacteria and archaea. They are roughly 100 times bigger, for one thing, and contain a nucleus—a saclike membrane that envelops their DNA.

Bacteria are familiar to us for the harm they cause our bodies—from cavities to tuberculosis. But the disease-causing species make up a tiny fraction of the full diversity of bacteria, which live everywhere from clouds to boiling deep-sea vents. Depending on the species, they may be shaped like rods, filaments, or spheres. Archaea are also single-celled organisms that can be shaped like rods, filaments, or spheres. They also live in a wide range of habitats. While archaea cannot carry out photosynthesis, they are capable of many other forms of metabolism. Archaea and bacteria may seem pretty much identical, but they have some important differences in their cells, such as the molecules that make up their membranes.

The dates marked on the tree of life indicate the ages of the earliest known evidence for each lineage. The earliest signs of life—such as the 3.45-billion-year-old stromatolites found by Allwood and her colleagues—strongly resemble living bacteria. In 2.6 billion-year-old rocks, scientists have found microbial fossils that bear a striking resemblance to one lineage of bacteria in particular: cyanobacteria, the bacteria that carry out photosynthesis. The age of these fossils is close to the oldest evidence of atmospheric oxygen that appears in the fossil record. The rise in oxygen was likely due to the emergence of cyanobacteria, which release oxygen during photosynthesis. While oxygen levels increased dramatically during this time, they were still very low compared to today. As a result, purple sulfur bacteria were still abundant 1.6 billion years ago, as reflected by the presence of okenane.

Archaea also make an early—but ambiguous—appearance in the fossil record. In 2006, Yuichiro Ueno and his colleagues at Tokyo Institute of Technology were able to extract methane from 3.5-billion-year-old rocks from Australia. The methane had a low fraction of carbon-13, indicating that it had been produced biologically (<u>Ueno et al. 2006</u>). Only one group of organisms alive today releases methane: a lineage of archaea called Euryarchaeota. Among the places they live today is the digestive tract of cows; they're the reasons that cow belches contain methane.

Eukaryotes emerge in the fossil record only about 1.8 billion years ago, with the appearance of single-celled organisms measuring about 100 micrometers across. Those early eukaryotes had ridges, plates, and other structures that are similar to those of living single-celled species. Over the next billion years, the diversity of single-celled eukaryotes increased as some lineages evolved to carry out photosynthesis while others preyed on bacteria or grazed on their photosynthetic relatives (Knoll et al. 2006).

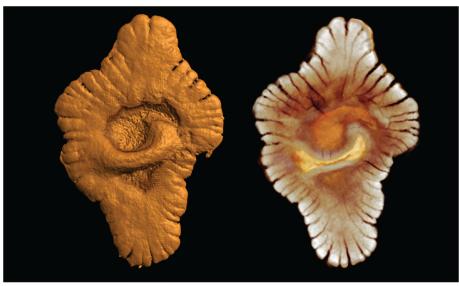
If you could travel back in time to 1.5 billion years ago, the world would appear to be a desolate place. On land there were no trees, no flowers, not even moss. In some spots, a thin varnish of single-celled organisms grew. In the ocean, there were no fish or lobsters or coral reefs. Yet the ocean teemed with microbial life, from the organisms that lived around hydrothermal vents on the seafloor to the free-floating bacteria and photosynthetic eukaryotes at the ocean's surface. Along the coasts, microbial mats stretched for miles in the shallow waters.

Today our attention may be distracted by animals and plants, but the world remains dominated by microbes. By weight, microbes make up the bulk of Earth's biomass. They live in a tremendous range of habitats that would kill the typical animal or plant—from Antarctic deserts to the bottom of acid-drenched mine shafts. The genetic variation among single-celled life also far exceeds that of animals or plants. Most genes on the planet belong to microbes or their viruses. It's a microbial world, in other words, and we just happen to live in it.

Life Gets Big

One of the most dramatic transitions in evolution was the origin of multicellular life. We need only look at our own bodies to appreciate this momentous change. Your body is composed of a trillion cells glued together with adhesive molecules and organized into tissues that cooperate intimately to keep the entire body alive. Only a minuscule fraction of cells in the human body—its sperm or eggs, depending on your sex—have the potential to pass on their genetic material to future generations.

The tree of life shows that multicellularity evolved dozens of times. The oldest evidence known from the fossil record—2.1-billion-year-old disks measuring up to 12 centimeters across—came to light in 2010 (Albani et al. 2010). While their shape appears biological, they don't look like any living form of life (FIGURE 3.10). The oldest recognizable multicellular eukaryotes—filaments of some type of algae—are 1.6 billion years old. The oldest known fossils of red algae date back 1.2 billion years (FIGURE 3.11), and green algae first appear 750 million years ago. Our own multicellular lineage—the animal kingdom—would not emerge until millions of years later.



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In 2010, scientists unveiled mysterious fossils measuring up to 12 centimeters across. Dating back 2.1 billion years, they appear to be the oldest fossils of multicellular life—but it's not clear what lineage they belonged to. (Information from <u>Albani et al. 2010</u>.)



Nicholas J. Butterfield.

FIGURE 3.11

A fossil of red algae, known as *Bangiomorpha*, dating back 1.2 billion years.

The Dawn of the Animal Kingdom

Animals are multicellular organisms that have to take in food to digest it. (Plants, by contrast, produce their own food by photosynthesis, while fungi release enzymes to break down food before absorbing it.) The animal kingdom today includes familiar groups such as mammals and birds, as well as less familiar groups such as sponges. Sponges may not seem much like us, since they lack a brain or eyes. But they share many key features with other animals, such as the fact that they eat. Instead of using a mouth to eat, they pump water through a network of tiny pores, where individual cells trap particles with long, hairlike structures. Sponges also share thousands of genetic elements only carried by animals, and not by other species.

Sponges appear to mark the earliest appearance of animals in the fossil record. In 2010, Adam Maloof of Princeton University and his colleagues reported 650-million-year-old fossils that appear strikingly like living sponges—down to spongelike pores (Maloof et al. 2010). Meanwhile, Gordon Love, a geochemist at the University of California at Riverside, and his colleagues have discovered sponge biomarkers of about the same age. In both Oman and Siberia, these researchers have isolated cholesterol-like molecules from 635-million-year-old rocks that are made only by a single group of sponges (FIGURE 3.12; Love et al. 2009; Kelly et al. 2011).



A: dsabo/Getty Images; B: Adam C. Maloof; C: Adam C. Maloof.

A: Sponges may belong to the oldest lineage of living animals. B: A 650-million-year-old fossil may belong to a sponge. The fossil is highlighted here in red. C: In this figure, the fossil is highlighted in a series of slices. If it is indeed a sponge, it would be the oldest known animal fossil. (Information from Maloof et al. 2010.)

The discovery of these biomarkers and fossils now gives us evidence that animals had already evolved at least 100 million years before the start of the Cambrian period. These ancient sponges were anchored to the seafloor. Some 50 million years later, the oldest signs of animals that can move appear in the fossil record.

Those moving animals didn't leave behind fossils of their bodies, however. Instead, they appear to have left behind their tracks. In Ecuador, Ernesto Pecoits of the University Alberto and his colleagues have found 585-million-year-old rocks with troughs in them that bear a striking resemblance to the tunnels made by burrowing worms today (FIGURE 3.13). Such animals would have been markedly different from sponges. They must have had muscles and nerves, for example (Pecoits et al. 2012).



Ernesto Pecoits.

Paleontologists recently found this fossil, and many others like it, in Ecuador. They concluded the marks were made by worm-like animals 2 to 3 millimeters in diameter. Dating back at least 585 million years, these fossils are the oldest evidence of animals that can move. (Pecoit et al. 2012)

As early as 575 million years ago, fossils of large animals turn up in the fossil record. Some looked like fronds, others like geometrical disks, and still others like blobs covered with tire tracks. Some fossils got as big as a meter across. These enigmatic species are known as the Ediacaran fauna, named for the Ediacara Hills, a region in Australia where paleontologists first recognized that these kinds of fossils dated back to before the Cambrian period (FIGURE 3.14).



Echo Medical Media.



Sinclair Stammers/Science Source.

Between 575 and 535 million years ago, a large diversity of multicellular organisms emerged. Known as the Ediacaran fauna, some of these species gave rise to living lineages of animals while others became entirely extinct.

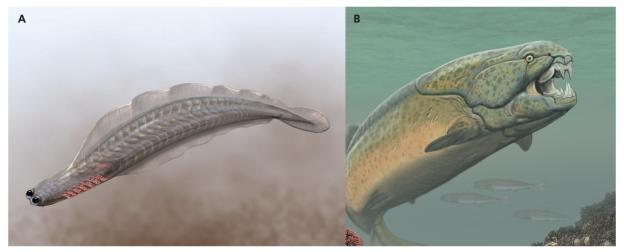
Some Ediacaran fossils share many traits with living groups of animals. *Kimberella*, for instance, has a rasp-shaped structure similar to what mollusks use today to eat. But many Ediacarans have proven far more difficult to decipher. Some fossils may be animals only distantly related to living lineages. Others may not be animals at all. They may have independently evolved multicellularity and then became extinct, leaving behind only their enigmatic fossils. (Xiao and Laflamme 2008).

Many Ediacaran lineages disappeared by the beginning of the Cambrian period, 542 million years ago, and they were completely gone by about 535 million years ago (FIGURE 3.15). In the meantime, some of the earliest recognizable members of living animal lineages had emerged (FIGURE 3.16; Erwin and Valentine 2013). We belong to the chordates, for example, a group that makes its first appearance in fossil-rich rocks in China called the Chenjiang Formation, which dates back 515 million years ago (Shu et al. 1999). In Chapter 10, we'll take a close look at the factors that were responsible for this remarkable unfolding of animal evolution.



Echo Medical Media.

The Ediacaran fauna was replaced by new groups of animals during the Cambrian period. This painting is a reconstruction of the 505-million-year-old ecosystem recorded in the Burgess Shale.



Carl Buell.

The earliest members of many living groups of animals first appeared during the Cambrian period. We humans belong to the chordate lineage that first appears in the fossil record during the Cambrian period and that gave rise to vertebrates. A: Haikouichthys was a small, fishlike animal with some traits found only in chordates, such as a brain and arches that may have supported gills. B: By 380 million years ago, large vertebrate predators had evolved, such as Dunkleosteus, which grew up to 6 meters long.

Climbing Ashore

Life took hold in the oceans, and it first climbed ashore billions of years later (Labandeira 2005). The oldest known signs of life on land are fossils of microbial mats from South Africa, dating to 2.6 billion years ago. Fungi, plants, and animals did not arrive on land until much later. In Oman, scientists have found 475-million-year-old fossils of spores that appear to have embedded originally in plant tissues—the oldest plant fossils found so far. The earliest land plants resemble mosses and liverworts (Wellman, Osterloff, and Mohiuddiu 2003). Over the next 100 million years, plants began to establish larger and larger ecosystems on land, until full-blown forests were growing (FIGURE 3.17).



Reprinted by permission from Macmillan Publishers Ltd:
Nature, "Giant cladoxylopsid trees resolve the enigma of the Earth's earliest forest stumps at Gilboa" © 2007 (reconstruction, left) Frank Mannolini, NY State Museum, Albany, NY; (photograph, right) South Mountain Trunk, William Stein, State University of NY at Binghamton, NY.

The oldest tree-like plant, known as *Wattieza*, was a 385-million-year-old plant that stood 8 meters tall.

Today, land plants live in intimate association with fungi. Some fungi feed on dead plants, helping to convert them into soil. Others cause diseases in plants, such as chestnut blight, which wiped out almost all American chestnut trees in the twentieth century. Still others help plants, supplying nutrients to their roots in exchange for organic carbon that the plants create in

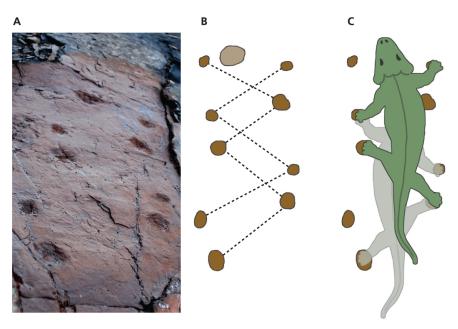
photosynthesis. The oldest fungus fossils, which date back to 400 million years ago, belong to this last category. Their fossils are mingled with the fossils of plants. It appears that fungi and plants helped each other move from water onto land (Berbee and Taylor 2007).

Animals left only tentative marks on the land at first. In rocks dating back to about 480 million years ago, there are tracks that appear to have been made by invertebrate animals—probably ancient relatives of insects and spiders. The tracks were made on a beach dune; whether the animal that made them could actually have lived full-time on land is a mystery. The oldest known fossil of a fully terrestrial animal is more than 50 million years younger than the first trackways: the fossil, a 428-million-year-old relative of today's millipedes, was found in Scotland in 2004 by a bus driver who hunts for fossils in his free time (FIGURE 3.18; Wilson and Anderson 2004). The oldest known trackways left by a vertebrate date back to 390 million years ago (Niedzwiedzki et al. 2010), while the oldest known fossils of vertebrates with legs—known as tetrapods—are about 370 million years old (FIGURES 3.19, 3.20). In the next chapter, we'll take a closer look at the fossil record of this last transition to land—the one that eventually gave rise to ourselves.



Carl Buell.

The oldest known fossil of a land animal belonged to a millipede dubbed *Pneumodesmus newmani*. (Information from Wilson and Anderson 2004.)



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: Grzegorz Niedzwiedzki.

FIGURE 3.19

A: In 2010, paleontologists reported a 390-million-year-old trackway made by vertebrates. B: The spacing of the tracks suggests they were made by an animal with an alternating gait (C).



Carl Buell.

Silvanerpeton was one of the oldest terrestrial vertebrates (or tetrapods).

Recent Arrivals

One of the most important lessons the fossil record teaches us is that the most familiar kinds of life today were latecomers. Most species of fish on Earth today, for example, belong to a group known as the teleosts. They include many of the most familiar fish, such as tuna, salmon, and goldfish. But 350 million years ago, there were no teleosts at all. Nor were there any mammals 350 million years ago, even though today they are the dominant vertebrates on land. Some 15,000 species of birds now fly overhead, but not a single bird existed back then.

Before today's most common groups of species emerged, the planet was dominated by unfamiliar ones. Before the rise of teleost fishes, for example, some of the ocean's top predators were giant sea scorpions, which measured up to 6 feet long. On land, 280 million years ago, the dominant vertebrates were ungainly, sprawling creatures called synapsids (FIGURE 3.21). The first synapsids that evolved into something that looked even remotely like today's mammals emerged about 200 million years ago. It was not until about 150 million years ago that the first members of the living groups of mammals evolved (Luo 2007).



Carl Buell.

Mammals are descended from sprawling, reptile-like vertebrates called synapsids that first emerged 320 million years ago.

Meanwhile, new lineages of reptiles were also evolving. One of the most successful was the dinosaur branch. Dinosaurs emerged about 230 million years ago and steadily grew more diverse. Their ranks included giant long-necked sauropods that were the largest animals ever to walk the Earth and fearsome predators besides. Dinosaurs dominated ecosystems on land until they disappeared in a pulse of mass extinctions 65 million years ago. The only survivors of this lineage today are the birds, which branched off from other dinosaurs about 150 million years ago (Chiappe 2007).

Most of the plants we see around us today are also relatively new in the history of life. As we saw earlier, the earliest fossils of plants resembled living mosses and liverworts. They likely formed low, ground-hugging carpets. The evolution of lignin and other tough plant compounds allowed some lineages to grow stems, stalks, and trunks. Starting in the Carboniferous period, several lineages of large plants began to appear. Many lineages later

became extinct, but some—such as ferns and ginkgo trees—have survived until today. They are no longer the dominant plant lineages, however. Today, most ecosystems are instead dominated by flowering plants. The oldest fossils of flowering plants date back to only 132 million years ago—some 300 million years younger than the oldest known plant fossils. During the Jurassic and Cretaceous periods, flowering plants became more abundant and diverse, possibly thanks to the simultaneous rise of beetles and other groups of insects that are now the most diverse (FIGURE 3.22).



NHPA/Superstock, inc.

FIGURE 3.22

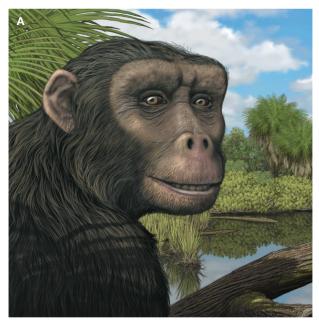
The oldest insect fossils are 400 million years old. But many of the most diverse groups of living insect species evolved much later. The first flies, for example, evolved about 250 million years ago. This fly (a gall midge) was trapped in amber about 30 million years ago.

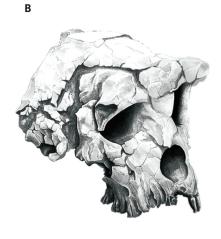
Today, one of the most widespread forms of flowering plants are grasses. Grasses cover the great expanses of savannas and prairies; they thrive in suburban lawns and city parks. The endless acres of wheat and corn that cover much of the world's arable land are nothing more than domesticated grasses. Yet grasses also arrive late in the fossil record, with the emergence of tiny bits of tissue preserved in 70-million-year-old dinosaur droppings. Grasses remained rare for many millions of years after that. Only about 20 million years ago did grasses expand into widespread territories.

The fossil record is filled with many examples of such long-term shifts in biological diversity, and, as we'll see in <u>Chapter 11</u>, a major goal of evolutionary biology is to test explanations for these changes. In the case of grasses, many researchers have argued that a gradual shift in the chemistry of the atmosphere has been responsible. Over the past 50 million years, carbon dioxide in the atmosphere has been declining gradually. Grasses are more efficient at extracting carbon dioxide from the atmosphere than are the shrubs that previously dominated many ecosystems (<u>Piperno and Sues 2005</u>; <u>Soltis et al. 2008</u>).

At the same time that flowering plants were emerging, the modern lineages of mammals were also becoming established. Not until the dinosaurs were gone did mammals begin evolving into dramatically new forms. Starting around 50 million years ago, for example, the ancestors of whales evolved from land mammals into the ocean's top predators (Chapter 1). At about the same time, bats evolved as the only flying mammals. The first fossils of primates are of the same age. The first primates were small, lemur-like creatures, but they shared many traits found in all living primates (including ourselves), such as forward-facing eyes and dexterous hands (Springer et al 2012).

Of all the living primates, our closest relatives are chimpanzees and bonobos. Species that are more closely related to us than to those apes are known as hominins. The oldest hominin fossils include *Sahelanthropus*, which was discovered in 2001 in Chad and dates back about 7 million years (FIGURE 3.23; Brunet et al. 2002). Early hominins were similar in some ways to chimpanzees, both in terms of their body and brain size. The oldest known fossils matching our own stature emerged only about 2 million years ago; and the oldest fossils that clearly belong to our own species, found in Ethiopia, are estimated to be nearly 200,000 years old (FIGURE 3.24; Johanson and Edgar 2006; McDougall, Brown, and Fleagle 2005; see page 52 for dating method).





(A) Carl Buell. (B) From "Two new Mio-Pliocene Chadian hominids enlighten Charles Darwin's 1871 prediction" by Michel Brunet. Philosophical Transactions of the Royal Society B: Biological Sciences, 27 October 2010: Vol. 365, No. 1556

A: A reconstruction of the oldest known bipedal hominid, *Sahelanthropus*. B: This fossil, discovered in the Sahara desert in 2001, is estimated to have lived 7 million years ago.



The Trustees of the Natural History Museum, London.

FIGURE 3.24

The oldest known fossil of our own species, discovered in Ethiopia, is estimated to be less than 200,000 years old. See <u>Box 3.1</u> for details about how scientists determined the age of this fossil.

Two hundred thousand years—the age of our species—is such a vast span of time that it's hard for the human mind to fathom. Ten thousand generations have passed since then. We're lucky if we meet our great-grandparents, three generations back. And yet, as we've seen in this chapter, 200,000 years is just a tiny fraction of the full span of time that life has existed on Earth. If you were to shrink all that time down to a day, our species would emerge 5 seconds before midnight.

The geological record of fossils, biomarkers, isotopes, and other traces of past life is clear evidence that life on Earth is immensely old. It also documents a profound transformation. For more than 1.5 billion years, the planet was inhabited solely by single-celled organisms. They were then joined on this planet by many multicellular life-forms. To understand how these sorts of changes occurred, scientists do not simply catalog lists of bones and stromatolites. They also figure out how different species—either alive today or long extinct—are related to one another. By determining these relationships, scientists can form hypotheses about the processes and patterns of evolution. How they discover life's kinship is the subject of the next chapter.

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How Biologists Use Phylogeny to Reconstruct the Deep Past

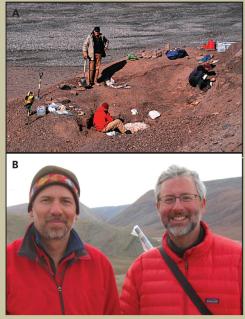


Carl Buell.

Tiktaalik lived 375 million years ago. It was an ancient relative of living tetrapods, which include amphibians, reptiles, and mammals. It had wrists and a neck, like living tetrapods, but it lacked other tetrapod traits such as true digits.

Neil Shubin spends the school year at the University of Chicago, where he teaches paleontology and anatomy. But his summers have frequently taken him north of the Arctic Circle, to a barren patch of land called Ellesmere Island. It's a harsh, dangerous place with so many hungry polar bears that Shubin and his colleagues all carry shotguns wherever they go. They spend so much time scanning the horizon for bears that their eyes play tricks on them. The scientists once saw what looked like a distant polar bear and scrambled for their guns, flares, and whistles. It took them a while to realize that the moving white blob was actually an Arctic hare, hopping along just a couple hundred yards away.

Shubin and his colleagues were drawn to this forbidding island because they wanted to find clues to one of life's major transitions. The polar bears, the Arctic hares, and Shubin himself are all tetrapods. Shubin wanted to learn about how tetrapods evolved from aquatic ancestors and how they went from living underwater to living on land. He decided to search for fossils of extinct species that evolved during this transition—species that might reveal details scientists could not find in living animals. Shubin and his colleagues read the scientific literature to figure out the likeliest locations for such fossils. Their research pointed them to a stretch of northern Canada that included Ellesmere Island.



Courtesy of Neil Shubin.

A: A team of scientists digging for fossils in the Arctic. B: Paleontologists Neil Shubin (*right*) and Ted Daeschler are part of the team that discovered a remarkable fossil they named *Tiktaalik*.

In 1999, Shubin and his colleagues flew to the island and began digging. They found fossils, but none were of early tetrapods. They had similar luck over the next three summers. By 2004, Shubin was wondering if it was time to bring the hunt to an end. But then, while cracking ice off some rocks in a lonely valley, he saw the outline of jaws that looked more like those of a tetrapod than those of a fish. The next day, his colleague Stephen Gatesy, a paleontologist from Brown University, found a second set of similar jaws. Gatesy could see that these bones were connected to a well-preserved skull. The skull was flattened, like the skulls of early tetrapods, and quite unlike the conical heads of fishes.

The scientists spent much of the summer of 2004 slowly excavating the rock where the bones were lodged. The fossil was flown by helicopter, and then by airplane, to Chicago. There, expert fossil preparators carefully picked away the rock, leaving the fossilized bones. The scientists had discovered a large portion of a skeleton of a truly remarkable creature that had lived 375 million years ago (Shubin 2008). It looked something like a fish with arms, and had the flattened head of a salamander. It measured about three feet from its flat head to its swimming tail. It had gills and scales, like a fish, but its front pair of appendages could bend at the elbow and could support the weight of its body. Shubin and his colleagues dubbed the creature *Tiktaalik roseae*. (*Tiktaalik* is the name of a fish in the Inuktitut language of northern Canada, and *roseae* honored one of the people who funded the expeditions to Ellesmere.)



Corbin17/Alamy Stock Image.

FIGURE 4.2

The discovery of *Tiktaalik* helped paleontologists understand how vertebrates moved from the water to land.

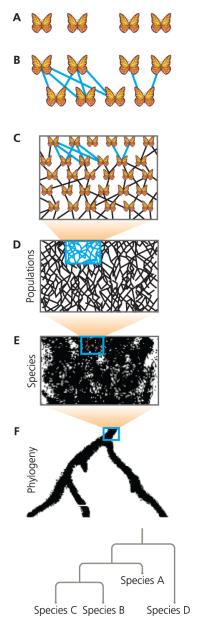
On its own, *Tiktaalik* would be just a very odd fossil. But it can tell us a great deal about our origins when we take into account one of Darwin's great insights: evolution produces new lineages like a tree growing new branches. Once Shubin and his colleagues were able to locate *Tiktaalik*'s branch on the tree of life, it spoke volumes

about the evolution of vertebrates that walk, fly, and jump around on land—including ourselves. And what is true for *Tiktaalik* is true for all species: to trace the spread of species across the planet, the origin of complex new traits, or even new diseases, scientists need to reconstruct the tree of life.

Tree Thinking

In the 1830s, Darwin began to picture evolution as a tree growing new branches (see <u>Figure 2.13</u>). In *The Origin of Species*, he put this image into words: "As buds give rise by growth to fresh buds, and these, if vigorous, branch out and overtop on all sides many a feebler branch, so by generation I believe it has been with the great Tree of Life, which fills with its dead and broken branches the crust of the earth, and covers the surface with its ever branching and beautiful ramifications" (<u>Darwin 1859</u>).

As scientists have come to better understand how evolution works, Darwin's metaphor has held up well. The new generations of a population are like the growing tip of a branch. If a population becomes divided, the subpopulations will continue to reproduce independently, like a branch splitting in two. These branches can split again and again, though extinctions can stop their growth (FIGURE 4.3). Scientists refer to the tree-like relationship of species to each other as phylogeny.

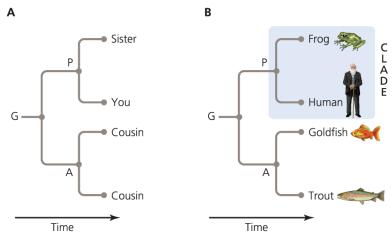


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The tree of life represents a hugely complex process. Organisms (A) mate to produce the next generation (B). C: Generation after generation, organisms pass down their DNA over vast stretches of time within a population (D). Interbreeding keeps populations linked together over time (E). New species arise when populations become divided and no longer interbreed (F). This splitting is akin to a branch splitting in two. As the isolated

populations continue to reproduce, the two new tips continue to grow. (Information from Baum and Smith 2012.)

Throughout this book, there are phylogenies that illustrate how species are related to each other. But to understand what a phylogeny actually shows you, it helps to think of a rough analogy (Gregory 2008). Let's say you have a sister and two cousins. You and your sister are more closely related to one another than either of you are to your cousins. So we can represent that kinship with a pair of branches joined to a node that represents a parent (FIGURE 4.4A). Your cousins are more closely related to each other than either of them is to you or your sister, so we can represent their relationship as another pair of branches joined together at a node of your aunt. And your parent and your aunt can, in turn, be joined to an older common ancestor: your grandparent.



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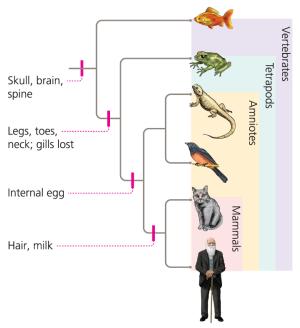
FIGURE 4.4

A: A phylogeny is similar in some ways to a genealogical tree. This tree shows the relationship between you, a sister, and two cousins. You and your sister share a common ancestor that your cousins do not (your parent). But you and your cousins also share a more distant ancestor (your grandparent). B: In a phylogeny, branches connect species instead of individuals. Humans and frogs share a closer common ancestor (an early tetrapod) that goldfish and trout do not share. But all four species also share a common ancestor—an early bony fish. (Information from <u>Gregory 2008</u>.)

The relationship of you to your relatives is similar in some ways to how species are related to one another. In <u>FIGURE 4.4B</u>, the four people at the tips of the branches have been replaced by four species. Humans and frogs share a common ancestry represented by branches and the node P. Likewise, goldfish and trout descend from a common ancestor A. And A and P, in turn, descend from a common ancestral species G. An organism and all its descendants are known as a clade, such as the one highlighted in <u>Figure 4.4B</u>.

The highlighted clade in turn is part of a larger clade—namely, G and all of its descendants, including goldfish and trout. In other words, one clade is nested inside another. This nesting pattern produced by evolution is what allowed Linnaeus to create his hierarchical classification system discussed in <u>Chapter 2</u>.

Linnaeus would identify a group of species because they all shared certain traits. All vertebrates have a skull and a spine, for example. FIGURE 4.5 shows a phylogeny of vertebrates. It shows how the hallmark traits of vertebrates evolved in their common ancestor and were then inherited by all of that ancestor's descendants. Only after the ancestors of goldfish (and all other ray-finned fishes) diverged from the ancestors of tetrapods did legs, digits, and other traits evolve. Those newer traits define a clade within the vertebrates: the tetrapods.

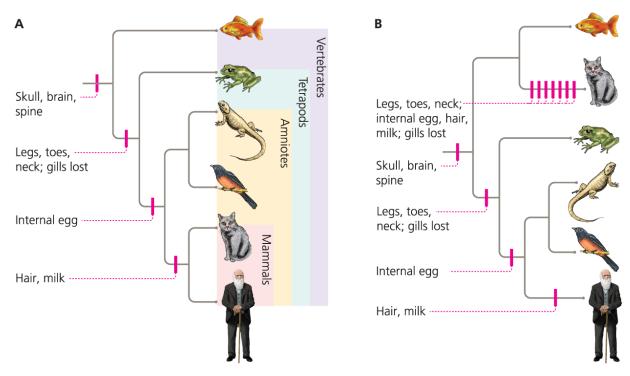


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Linnaeus classified species into groups nested within larger groups. The branching process of evolution makes it possible to do so. All vertebrates have a skull, brain, and spine, which they inherited from a common ancestor. Mammals, which form a clade within the vertebrate clade, have additional traits not found in other vertebrates, such as hair and milk.

Uncovering the Tree of Life

To uncover the branches of the tree of life, biologists compare different organisms to identify the traits they share. Let's say that you wanted to figure out how people, cats, birds, lizards, frogs, and goldfish are related to each other. You could draw 945 different trees that could connect these six animals. FIGURE 4.6 shows two of them. In one, cats and humans form one clade. In the other, cats and goldfish do.



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FIGURE 4.6

A: This tree is a well-supported hypothesis for how six vertebrate species evolved from a common ancestor. The dashes show when the ancestors of some of these species first evolved unique traits they share. Biologists use these shared traits to name groups of species (such as mammals). While this tree includes only a few traits found in vertebrates, a vast number of other traits support this tree. B: Biologists test alternative trees to see how well they can explain the evidence. If cats were more closely related to

goldfish than to humans, they would have to independently evolve a large number of traits.

Each tree is a scientific hypothesis, but both can't be right. Biologists can evaluate them using a number of different methods: Some hypotheses explain the data more simply than others. If vertebrates had evolved to produce the pattern in Figure 4.6B, for example, cats would have needed to independently evolve many traits on their own. They would have independently evolved legs and digits like those of tetrapods, internal fertilization like that of amniotes, hair like that of mammals, and so on. The hypothesis that cats are mammals is a much simpler hypothesis involving fewer steps (Baum 2012).

This is not to say that two lineages cannot independently evolve the same trait—in a process known as convergent evolution. Birds, bats, and beetles, for instance, all have wings they use for powered flight (FIGURE 4.7). When scientists reconstruct a phylogeny, they must take special care to avoid confusing homologous traits with convergent ones. If we take a close look at wings, for example, we can see that their superficial similarities hide fundamental differences. Bird wings have feathers, whereas bats have skin stretched between their digits. Bird and bat wings both contain internal bones, while beetle wings are constructed from entirely different tissues. By comparing species at this level of detail, biologists can see how each lineage acquired wings.



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Birds and insects both can fly with wings. But birds and insects didn't inherit their wings from a winged ancestor. In other words, their wings are not homologous. They evolved them separately, which is why bird wings are made of bone and muscle while insect wings are made from an exo-skeleton. This independent evolution is known as convergence.

Once scientists reconstruct a phylogeny, they can use it to explore questions about evolution. Here we'll use phylogenies to tackle four particularly intriguing questions: How did life come on land? Where did our ears come from? How did birds evolve flight? And, finally, how did our ancestors stand upright?

From Fins to Limbs: Homology through Time

The evolutionary tree in <u>Figure 4.6</u> has five tetrapods and one fish. The difference between the two groups is obvious. All the tetrapods have limbs with digits that they use to move around on land. The goldfish, by contrast, has fins it uses to swim underwater. The tissues inside its fin are also dramatically different from the ones inside the tetrapod limb. Tetrapod limbs have the same basic arrangement of skeletal bones—a long bone close to the body, two more rod-shaped bones further out, a set of small roundish bones, and finally a set of digits. A goldfish fin consists of a small fan of skeletal bones surrounded by fin rays, an entirely different kind of tissue.

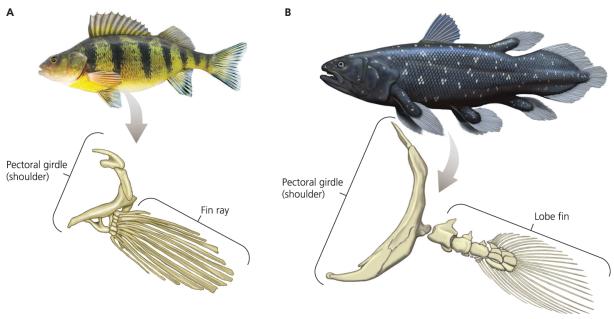
So where did the tetrapod "body plan" come from? That's the question that propelled Neil Shubin and his colleagues to the Arctic Circle. And evolutionary trees guided them there.

Studies on fossils and on the anatomy of living vertebrates have revealed the closest aquatic relatives of tetrapods. Their ranks include coelacanths, stout-bodied fishes that live in the deep sea off the eastern coast of Africa and the waters around Indonesia (FIGURE 4.8). Another group of tetrapod relatives are lungfishes, a group of species that live in rivers and ponds in Brazil, Africa, and Australia. Coelacanths and lungfishes don't have the familiar webbed fins that you might find on a goldfish or a trout. Instead, their fins are fleshy lobes that contain stout bones. As a result, they're known collectively as lobe-fins (FIGURE 4.9).



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Coelacanths are large fish that live deep underwater in the Indian Ocean. They are among the closest living relatives of tetrapods.



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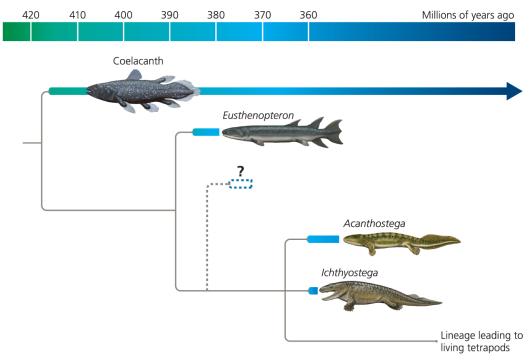
FIGURE 4.9

A: the ray-finned fishes, known as teleosts, have fins that are made up mostly of slender rays. B: Coelacanths, on the other hand, have a short chain of bones that anchor powerful muscles. This appendage is homologous to the tetrapod limb. Our own limbs evolved from a related "lobe-fin."

The common ancestors of tetrapods and lobe-fins lived some 400 million years ago. After they split, each lineage evolved into dramatically different forms. Living lungfishes have since adapted to living in freshwater, while coelacanths have adapted to the deep ocean. The earliest branches of the tetrapod clade have all become extinct. And so the only place to find more insight into the origin of tetrapods is in the fossil record.

Paleontologists found the first of these fossils in the late 1800s. *Eusthenopteron*, which lived about 385 million years ago, had a stout bone extending from its shoulder girdle and two more bones extending further out. Over the course of the 1900s, a few more transitional fossils emerged. In Greenland, for example, Jennifer Clack of the University of Cambridge found the remains of a 365-million-year-old tetrapod called *Acanthostega*. It had complete tetrapod limbs, including eight toes on each foot. But it also had many ancestral traits found in *Eusthenopteron* and other lobe-fins. Its skeleton was not adapted for supporting its body on land, for example, and it had bones for supporting gills (Clack 2002).

In the 1990s, Neil Shubin and his colleagues joined the hunt for early tetrapods. Shubin was at the University of Pennsylvania at the time, and so the team started nearby, in the Appalachian Mountains. There, they found an isolated shoulder bone from an early tetrapod that lived some 360 million years ago. Encouraged by the discovery, they began researching other geological formations they might visit to find more tetrapod fossils. The phylogeny of lobe-fins known at the time allowed them to generate a hypothesis about where to go (FIGURE 4.10).

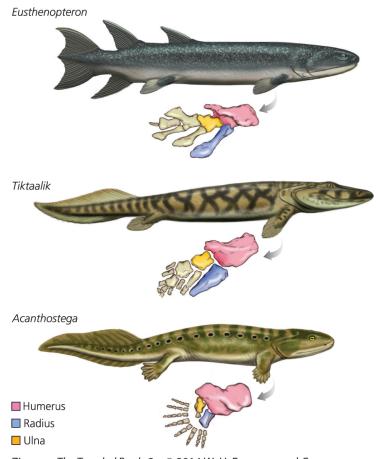


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Phylogenies allow scientists to come up with hypotheses that can drive new research. Phylogenies generate hypotheses. This phylogeny represents what scientists understood about the origin of tetrapods around the year 2000. Early tetrapods and closely related lobe-fins lived in the mid-Devonian. Neil Shubin and his colleagues hypothesized that transitional species would be found in mid-Devonian rocks. This hypothesis led them to their discovery of *Tiktaalik*.

Up till then, all of the fossils marking this transition had been found in formations dating back to the mid-Devonian period, from about 370 to 350 million years ago. Shubin and his colleagues reasoned that it was the best time period in which to search for other early tetrapods. Paleontologists had found early tetrapods and their closest lobe-fin relatives in rocks that had formed in coastal wetlands and river deltas. And so Shubin and his colleagues narrowed their search further, to mid-Devonian sedimentary rocks that had formed in those environments. While examining a stratigraphic map, they noticed a swath of mid-Devonian sedimentary rocks in northern Canada that had yet to be explored for tetrapods. They decided to head north.

Just as Shubin had hoped, those rocks revealed a lobe-fin that was more closely related to living tetrapods than *Eusthenopteron*, but less so than *Acanthostega*. It had long limb bones as well as small bones corresponding to those in our own wrists (FIGURE 4.11). This lobe-fin, *Tiktaalik*, also had a neck—something that *Eusthenopteron* and other lobe-fins lack. But it did not have digits, like *Acanthostega* did. *Tiktaalik* had precisely the mix of ancient and new traits that would be expected of a creature that occupied a branch between lobe-fins and the tetrapods that evolved from them. Shubin's discovery of *Tiktaalik* thus shows how paleontologists can make predictions from hypotheses, even if they're studying events that took place hundreds of millions of years ago.



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FIGURE 4.11

Lobe-fins and early tetrapods have homologous bones in their limbs. *Eusthenopteron* had bones that were homologous to the long bones (the humerus, radius, and ulna) of our

arms. *Tiktaalik* had additional homologies, including wrist bones. *Acanthostega*, an early tetrapod, had distinct digits at the ends of its limbs. While all tetrapods today have only five or fewer digits, *Acanthostega* had eight. (The wrist is absent from *Acanthostega* in this figure because the bones have not yet been discovered. Information from <u>Friedman</u>, <u>Coates, and Anderson 2007</u>.)

FIGURE 4.12 shows the phylogeny of lobe-fins based on an analysis of *Tiktaalik*, other Devonian fossils, and living species. This figure, known as an evogram, has more detail than the cladogram in Figure 4.6. For one thing, the branches are arranged along a timeline. The thick blue lines represent fossils, whose ages we know, and the gray lines represent their lineages. The age of the nodes are only estimates, based on the known ages of the fossils. It also shows how some key traits changed as the tetrapod body plan evolved from that of their lobe-fin ancestors.

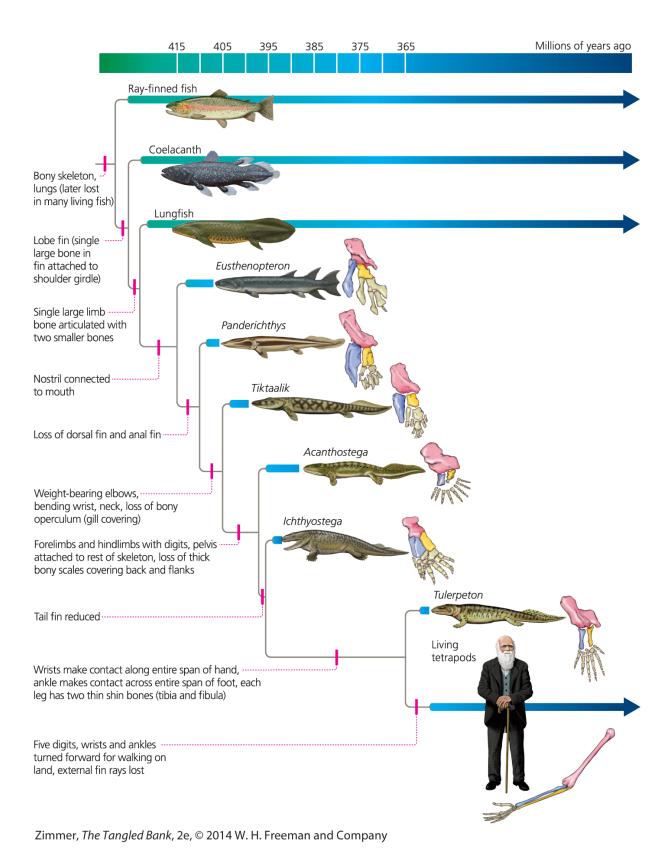


FIGURE 4.12

This tree shows the relationship of lobe-fins and tetrapods. The tetrapod "body plan" evolved gradually, over perhaps 40 million years. This tree includes only a few representative species; paleontologists have discovered many others that provide even more detail about this transition from sea to land. A significant insight from this phylogeny is that much of the tetrapod body plan, including limbs and digits, evolved while tetrapods were still aquatic.

Bear in mind that the species in Figure 4.12 do not form a continuous line of ancestors and descendants. *Tiktaalik* has some features that aren't shared by other lobe-fins or tetrapods, but are instead unique to *Tiktaalik* alone. These peculiar traits probably evolved after its ancestors diverged from the ancestors of other lobe-fins. Still, this tree gives us insights about the evolution of tetrapods that we'd never have if not for fossils. It shows us that the common ancestor of tetrapods and their closest living relatives had stout, paddle-shaped fins, for example. *Tiktaalik* probably could have pushed up its head and shoulders, judging from its bones and the attachments for muscles. Digits evolved in the common ancestor of *Acanthostega* and other tetrapods. *Acanthostega* had eight digits; other species had six or seven. All living land vertebrates have five or less. Because of fossils, we now know that this five-finger rule took millions of years to emerge. (Daeshcler, Shubin, and Jenkins 2006; Shubin, Edward, and Farish 2006).

This evolutionary tree also lets us test hypotheses about the selective pressures that led to the origin of the tetrapod body plan. In the early 1900s, Alfred Romer developed an influential theory based on the knowledge that the oldest tetrapods at the time were found in rocks that appeared to have formed during a time of severe droughts. He envisioned fishlike vertebrates living in rivers and ponds; when they dried up, the animals had to make their way to remaining bodies of water or die. Mutations that led to more leg-like fins would enable them to move faster over land.

The phylogeny in <u>Figure 4.12</u> falsifies this hypothesis. Even after tetrapods had evolved fully formed tetrapod limbs, they were poorly suited for life on land. *Acanthostega*, for example, had limbs and digits, but it also had bones for supporting gills that it may have used to get oxygen. Its shoulder and pelvic bones were so slender that they probably couldn't have supported its

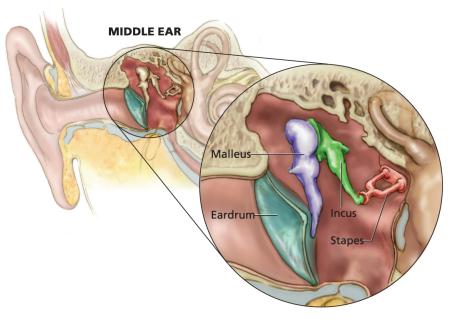
weight on land. *Acanthostega's* tail remained lined with delicate fin rays that would have been damaged if the animal dragged its tail along the ground.

These early tetrapods did not live in a harsh, arid landscape as Romer envisioned; instead, they lived in lush coastal wetlands. Shubin and other paleontologists have proposed that early tetrapods used their legs to move underwater, perhaps holding onto underwater vegetation or clambering over submerged rocks. It is not until millions of years later that fully terrestrial tetrapods emerge in the fossil record.

Evolution as Tinkering

In 1977, the French biologist and Nobel Prize winner François Jacob published an influential essay entitled "Evolution and Tinkering" (<u>Jacob 1977</u>). "In contrast to the engineer," he wrote, "evolution does not produce innovations from scratch. It works on what already exists, either transforming a system to give it a new function or combining several systems to produce a more complex one." Jacob likened evolution to a tinkerer who makes little changes to things that already exist.

Sometimes it's hard to understand how tinkering can produce life's complexity. Phylogenies can help reveal this process. Our ears, for example, contain a delicate chain of bones. When struck by sound waves, these bones transmit the vibrations to nerve cells. All living mammals have this combination of ear bones, but the closest living relatives of mammals, such as birds and lizards, lack it (FIGURE 4.13). Thanks to the fossils that paleontologists have discovered over the past few decades, we know that the mammalian ear didn't simply pop out of nowhere. The bones we use to hear once helped our ancestors to bite.

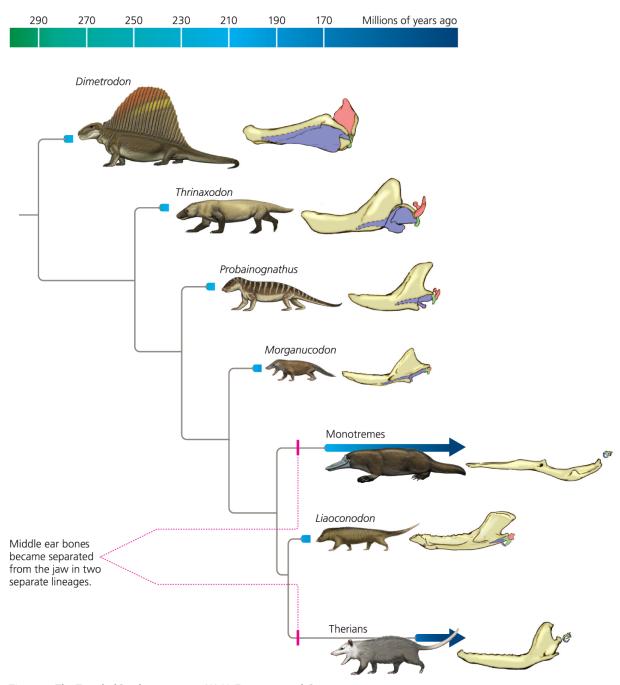


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Humans and all other living mammals have three small bones in the middle ear that transmit sound from the eardrum to the inner ear. These bones develop in mammalian embryos as part of the lower jaw. Living reptiles and birds have no such structure in their ears, raising the question of how the mammalian ear evolved.

As we discussed in <u>Chapter 3</u>, mammals belong to a clade of tetrapods known as synapsids. The earliest synapsids didn't look much like today's cats, humans, or any of the other hairy creatures we're familiar with. *Dimetrodon* had a strange, sail-shaped back. Others looked like turtles with fangs. Nevertheless, all synapsids shared certain features in their skeletons—particularly the way their skull bones fit together—that are found today only among mammals. By studying these features, scientists can trace the emergence of mammals from their reptilian forebears.

About 260 million years ago, a lineage of synapsids known as the cynodonts emerged, displaying new mammal-like traits such as a more upright stance. By about 200 million years ago, the basic mammal body plan known today had evolved, and it is seen in fossils of such mammals as *Morganucodon* (FIGURE 4.14; Luo 2007).



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The phylogeny of mammals and their extinct relatives makes it possible to trace the origin of the middle ear. The ancestors of mammals had lower jaws made up of several large bones. Over millions of years, three bones at the rear of the jaw shrank and separated, becoming specialized for transmitting sounds in the ear. Their homology is indicated here by their matching colors. Fossils discovered in recent years have

revealed that ear bones separated independently in the ancestors of the two main lineages of living mammals, the monotremes and the therians (a group that includes marsupial and placental mammals).

There are three living branches of mammals. Monotremes include the duckbilled platypus and the echidna. They produce milk, but only through a loose network of glands rather than through a nipple. Like reptiles and birds, they lay eggs. All the remaining species of living mammals bear live young. These mammals, known as therians, form the two other branches. One branch, the marsupials, includes opossums, kangaroos, and the koala. Marsupial young crawl into a pouch on the mother's belly after they're born, where they can be carried until they're big enough to survive on their own. The other branch, the eutherian mammals, includes humans and all other mammals that develop a placenta to feed embryos in the uterus.

Figure 4.14 shows the phylogeny of mammals, highlighting the evolution of the ear. It shows how the bones of the middle ear started out as part of the lower jaw. In early synapsids, the foremost bone in the jaw, the dentary, held many teeth, and the bones in the rear formed a hinge against the back of the skull. Like many reptiles today, early synapsids had simple ears. They may have picked up vibrations through their jaws that were then relayed back to the middle ear.

In the mammal lineage, the dentary gradually became larger and larger. Larger dentaries may have been favored by natural selection because they provided more strength than a group of smaller bones. As the dentary expanded, the bones in the back of the jaw shrank. At first, these shrinking bones continued to help anchor the lower jaw. Later, the dentary became the sole bone of the jaw, making direct contact with the back of the skull.

During this transformation, as fossils show, some of the shrinking bones of the rear lower jaw disappeared entirely. But others took on a new role. They were co-opted into the network of bones in the ear. These former jawbones made the mammalian ear a better listening organ, serving as levers that amplified faint high-frequency sounds. At first, the fossils show, this chain of bones remains tethered to the lower jaw. In the ancestors of the living mammals, the chain later became free of the attachment.

As paleontologists discover new fossils, our understanding of the origins of the mammal ear gets better. In 2011, for example, a team of Chinese and American scientists published the details of a skeleton belonging to a 125-million-year-old therian fossil called *Liaoconodon hui* (Meng, Wang, and Li 2011). *Liaoconodon* was more closely related to living therians than to monotremes. Yet the bones of its middle ear still retained a connection to the lower jaw. Adding a branch for *Liaoconodon* reveals a surprising twist to the history of the mammal ear. The bones of the middle ear separated from the lower jaw independently in the monotreme lineage and in the therian lineage.

Like the other evograms in this chapter, <u>Figure 4.14</u> only illustrates the phylogeny of a small portion of the fossil mammals paleontologists have found. Those fossils have revealed that the middle ear evolved independently in other lineages as well—lineages that later became extinct. Evolution's tinkering, it turns out, has been an exuberant experimentation.

Feathered Dinosaurs Take Flight

Today, some 15,000 species of birds share a remarkable trait found in no other animal: feathers. Feathers are exquisitely adapted for flight; despite being practically weightless, they can lift a bird hundreds of feet into the air and take it from one side of the Earth to the other. The phylogeny of birds and their closest relatives demonstrates that feathers have a surprising evolutionary history: the feather came before the flight.

In 1860, just a year after *The Origin of Species* was published, German quarry workers discovered the fossil of a bird like nothing alive today (FIGURE 4.15). It was clearly a bird, with a bird's skeleton and a bird's feathers. (The impressions of the feathers were preserved thanks to the still, oxygen-free swamp the bird fell into when it died.) But the fossil, which turned out to be 145 million years old, also had teeth in its beak, claws on its wings, and a long, reptilian tail. Scientists dubbed it *Archaeopteryx*, "ancient wing" (Shipman 1998).



(A) imageBROKER /Superstock, Inc. (B) Carl Buell.

A: In 1860, just after Darwin published *The Origin of Species*, German quarry workers discovered a fossil of a bird with reptile traits such as teeth and claws on its hands. Known as *Archaeopteryx*, it lived 145 million years ago. B: A reconstruction of what *Archaeopteryx* may have looked like.

Before the discovery of *Archaeopteryx*, birds seemed profoundly different from all other living things. They shared many unique traits, such as feathers and fused arm bones not found in other tetrapods. *Archaeopteryx* offered clues to how birds had evolved from reptile ancestors. But *Archaeopteryx* alone left many questions unanswered. Did feathers evolve first, before flight? Which reptile ancestor did birds evolve from? For a century, those questions remained open. But over the past 40 years, thanks to new fossil discoveries and careful comparisons of fossils and birds, a consensus has emerged. Birds are living dinosaurs (<u>Chiappe 2007</u>).

Box 4.1

Who Are You Calling "Primitive"?

or millennia, Western naturalists organized living things into "lower" and "higher" forms, envisioning life arrayed along a ladder-like scale. We now know this is not true. A tree is a better metaphor for life than a ladder. Everything alive today, from bacteria to jelly fish to humans, belongs to the same 3.5-billion-year-old lineage, and so there is nothing intrinsically superior about animals, let alone humans. Indeed, if we want to judge success by numbers alone, the most successful form of life is the humble virus, which has an estimated population of 10³¹ (Zimmer 2011).

Yet it's hard to shake the old "ladder thinking." Take a look at the mammal tree on <u>page</u> 85. It's tempting to see it as a progress from synapsids to monotremes like platypuses to therians like ourselves. The biology of platypuses can add to this misunderstanding. They reproduce by laying eggs, for example, like reptiles and amphibians. Only later did the ancestors of therians evolve embryos that developed in their uterus.

In fact, platypuses are not lower than we are, nor are they primitive. It is certainly true that platypuses have some of the traits that the ancestors of therians had. But those ancestral traits of platypuses are only part of their biology. Platypuses have evolved a

unique bill, for example, which they sweep through water to detect electricity from their prey. Platypuses also have webbing on their feet and produce venom.

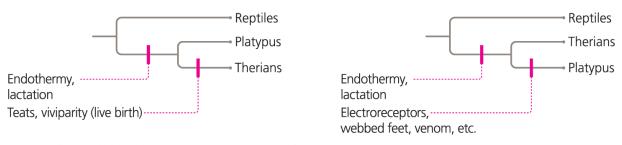
These remarkable traits evolved only *after* the ancestors of platypuses split off from living mammals. And scientists will probably add many more traits to this list. In 2008, an international team of scientists sequenced the platypus genome and found that it contained thousands of genetic elements that did not evolve until after their split from other animals (Warren et al. 2008).

Biologists can gain clues to the nature of early mammals by comparing platypuses to other mammals. But we cannot treat living platypuses as a stand-in for that ancestor. Indeed, when it comes to electroreceptors, bills, and venom, we humans are the primitive mammals (Omland, Cook, and Crisp 2008).



age fotostock/Superstock, Inc.

FIGURE 1 Platypuses are mammals that lay eggs. That does not mean they are "primitive," however.



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FIGURE 2 For some traits, platypuses still have the ancestral form, which has been modified in therians. But platypuses also have new traits not found in therian mammals. We could rearrange the same tree, mark a different set of characters, and give the impression that it is humans who are "living fossils."

FIGURE 4.16 shows how birds are related to dinosaurs and other reptiles. In the 1970s and 1980s, paleontologists observed that the skeletons of birds share many traits with those of one group of dinosaurs in particular, a group known as the theropods. Theropods were bipedal meat-eating dinosaurs whose ranks included *Tyrannosaurus rex* and *Velociraptor*.

Living birds

digits, short feathered tail Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company

Long arms (ulna longer than femur) -----

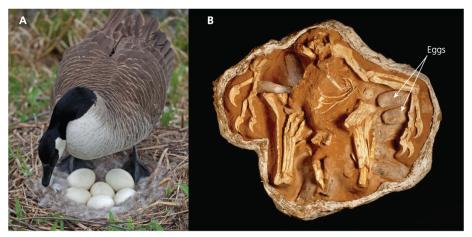
bird-like sleeping position

Toothless beak, fused wing

All birds have feathers, but feathers evolved long before birds. As this phylogeny indicates, early ground-running dinosaurs already had simple feathers, which became more complex before they were used for powered flight.

In the late 1990s, a new trait emerged to link theropods and birds: feathers. New fossils, mostly found in China, revealed ground-running theropods that were covered with strange outgrowths. Close analysis of these structures revealed some features that are found today only on feathers, such as vanes, barbs, and a central stalk. As we saw on page 54 in Chapter 3, these feather fossils sometimes preserve melanosomes, which offer clues to the colors of the feathers (Li et al. 2010).

In other words, the traits of birds began to evolve long before birds existed. Early theropods obviously couldn't have used feathers for flight, because their arms were too short and their feathers couldn't lift them off the ground. But birds also use feathers for other things, such as offering a way for other members of their species to recognize them, and also for attracting mates. The finding that even the early, bristle-like feathers already had colorgenerating melanosomes supports the hypothesis that early dinosaurs used their feathers as visual signals. The dinosaurs most distantly related to birds in the phylogeny in <u>Figure 4.16</u> had only bristle-like feathers; in other lineages, feathers became more elaborate, although the dinosaurs still couldn't fly. It's possible that dinosaurs used these intermediate feathers for insulation or incubating eggs. This last possibility is particularly striking when you consider a fossil discovered in 1993 (Norell et al. 1995). A theropod known as an oviraptor lies over its nest of eggs, its arms spread out in much the same way modern birds spread their wings to protect their nests (FIGURE 4.17).



(A) John Cancalosi/Oxford Scientific/Getty Images. (B) Mick Ellison/American Museum of Natural History.

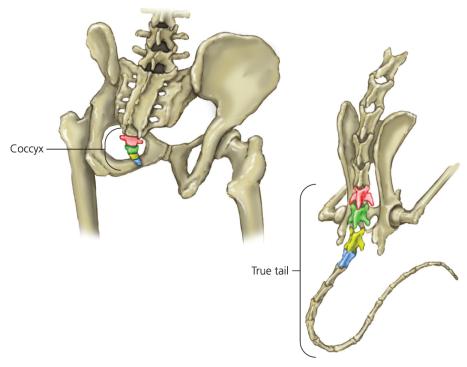
Dinosaur fossils reveal the ancient history not just of the anatomy of birds, but of their behavior. A: Birds incubate their eggs by sitting on them to keep them warm. B: In Mongolia, paleontologists have discovered nesting fossils of dinosaurs in a similar posture. This discovery suggests the nesting behavior seen today in living birds evolved over 150 million years ago in non-flying feathered dinosaurs.

Because these feathered dinosaurs are the closest known relatives of birds, scientists can study them to make hypotheses about how flight evolved. Ken Dial of the University of Montana, for example, has discovered that in many species, young birds that cannot fly still flap their wings (Dial, Jackson, and Segre 2008). Even a small covering of feathers allows them to generate a downward force that gives them extra traction while running up inclines. It's possible that some feathered theropods evolved a flight stroke even before they could fly, to help them run from predators or capture prey. And in one lineage of small, feathered dinosaurs, this speed-boosting flapping apparently evolved into true flight.

A New Ape

When Charles Darwin wrote *The Origin of Species*, he said next to nothing about the evolution of humans. "Light will be thrown on the origin of man and his history" was about all he had to say (<u>Darwin 1859</u>). We know why: In a letter to a friend, Darwin wrote that delving deeply into human evolution would prejudice readers against his theory. It was one thing to say that whales had evolved from bear-like terrestrial mammals. It was quite another to say that humans were related to apes. Despite his public quietness, Darwin gave human evolution a great deal of thought. In 1871 he felt the time was right to share those thoughts, which he did in a book called *The Descent of Man*.

In his book, Darwin argued that the anatomy of the human body bore witness to its evolution. Humans have vestigial features, for example, such as a stump of a tail (FIGURE 4.18). Some babies are even born with the stump of a tail emerging from their backs. Such a feature would seem arbitrary if humans were specially created, but it would make ample sense if our ancestors once had tails. Among the primates, Darwin argued, the closest living relatives to humans were the African apes. (Darwin knew of gorillas and chimpanzees, but today scientists recognize a third African species, the bonobos, which had a common ancestor with chimpanzees about 2 million years ago [Bjork et al. 2011].) Darwin noted many similarities between humans and apes, from the details of our skeletons to our similar facial expressions.



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The base of the human spine corresponds, bone for bone, to the base of a monkey's tail. The best explanation for this homology is that we descend from a common ancestor that had a tail. The ancestor of humans and other apes lost a fullblown tail but retained its vestiges. (Information from Coyne 2009.)

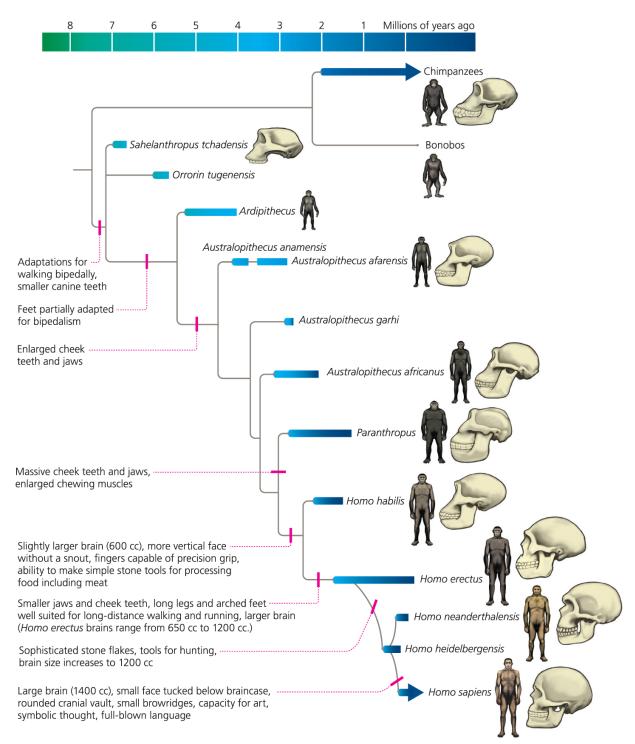
At some point after our ancestors split from those of other apes, Darwin argued, they must have evolved to walk upright. But Darwin had no evidence for testing this hypothesis. Only one fossil hominin had been discovered at the time—what came to be known as the Neanderthal. And it wasn't even clear if Neanderthals were a separate species or just a population of humans with some unusual features, such as thick browridges. So Darwin's hypothesis generated a prediction: in the future, paleontologists might find fossils of species that had evolved some of the traits found today only in humans. Those species also might have retained some of the traits shared by other apes, but lost in humans.

By the early 1900s, paleontologists were finding fossils that met Darwin's prediction. In 1924, a South African physician named Raymond Dart

identified the skull of a child with forward-facing eyes and a small jaw, much like humans have. But the skull also had many traits that linked it with apes, such as a small braincase. And since then, scientists have identified the fossils of some 20 different kinds of hominins.

The subject of human evolution is stupendously rich. <u>Chapter 14</u> will focus entirely on our origins, and human evolution will make appearances from time to time before then. But in this chapter on phylogeny, we'll limit ourselves to considering just the phylogeny of hominins and what it tells us about the evolution of the human body.

There's a spirited debate these days about some aspects of hominin phylogeny. Paleoanthropologists argue over whether certain fossils represent separate hominin species or are merely variations of a single species, for example. Some researchers maintain that hominins have frequently experienced convergent evolution, making it difficult to figure out where some lineages actually fit in the hominin family tree. Others think that convergence was rarer. Fortunately, these are all hypotheses that scientists can test with the discovery of new fossils. And while paleoanthropologists disagree about some points of human evolution, they've formed a consensus about others (FIGURE 4.19).



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FIGURE 4.19

This tree shows the relationship of humans to living apes and extinct hominins. The earliest known fossils of hominins suggest that our ancestors were partially bipedal 7

million years ago. This is a tentative conclusion, because paleontologists have found only a few fossils from this age. More consistent evidence for bipedality emerges in hominins that lived between 4 and 3 million years ago. Later hominins evolved large brains and sophisticated tools. This phylogeny shows only a small selection of the 20 species of hominins known (Strait, Grine, and Freagle 2007; see also Johanson and Edgar 2006 and Andrews 2005). (Information from Strait et al. 2007.)

The fact that chimpanzees, bonobos, and gorillas are the closest living relatives of humans offers some evidence about what the first hominins were like. All of these apes have small brains, compared with those of humans, so it's likely the first hominins did, too. Because these living apes all walk mainly on both their feet and hands, the earliest hominins likely inherited this way of walking as well.

The earliest fossils of hominins date back to a time between 6 and 7.5 million years ago. By that time, hominins had already evolved a few traits that set them apart from other apes. For example, a hominin called *Sahelanthropus tchadensis* had smaller canine teeth than other apes, and its cheek teeth were thicker. Another difference was where the spinal cord exits the back of the skull, through a hole known as the foramen magnum. In chimpanzees and other apes, this hole is oriented toward the animal's back. In *Sahelanthropus*, the hole was oriented downward, much like the human foramen magnum. This suggests that the head of *Sahelanthropus* sat atop its neck like ours, rather than extending forward like a chimpanzee's (<u>Brunet et al. 2005</u>).

This kind of stance would be consistent with standing on two legs rather than on four. Unfortunately, when paleontologists discovered *Sahelanthropus* in Chad in 2001, they found only parts of its skull and jaw. They have yet to find bones from the rest of its body. But other researchers have found leg bones in Kenya from a 6-million-year-old hominin called *Orrorin tugenensis* (Richmond and Jungers 2008). *Orrorin's* thighbone (the femur) had a ball at its top that was oriented much like the ball on the femur of a modern human. It's possible that this arrangement was an adaptation for bearing the weight of an upright upper body (FIGURE 4.20).



Brian G. Richmond.

FIGURE 4.20

Orrorin tugenensis was a hominin that lived in Kenya 6 million years ago. Its femur, shown here, appears to have been adapted for supporting an upright torso. This bone suggests that *Orrorin* and other early hominins were walking at least partially upright.

In 2009, a team of Ethiopian and American scientists published a detailed report on an exciting discovery about early hominins (White et al. 2009). They actually made the discovery 15 years earlier—a 4.4-million-year-old fossil they named *Ardipithecus ramidus*—but they then had to painstakingly prepare and analyze the 110 pieces of its skeleton they unearthed. When they were done, they had a remarkable glimpse at a large portion of an early hominin's body as well as the environment in which it had lived. FIGURE 4.21 is a reconstruction based on their work. *Ardipithecus ramidus* still had a small head and large jaw. But it also had some traits that were signs of bipedality. On its pelvis, for example, it had some of the same anchors for muscles that we have but chimpanzees and other apes do not.



(A) David L. Brill. (B) Carl Buell.

FIGURE 4.21

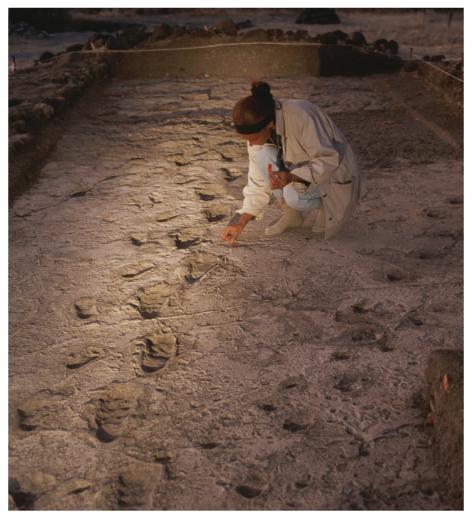
A: A relatively complete skeleton of *Ardipithecus ramidus*, which lived 4.4 million years ago. B: *A. ramidus* shows signs of being adapted for both bipedal walking and arboreal life.

On its feet, *Ardipithecus* had four short little toes that were kept stiff, perhaps allowing it to walk more efficiently on the ground than earlier hominins had. But its big toe was still opposable, much like our thumbs. This sort of big toe probably helped *Ardipithecus* move through the trees. But *Ardipithecus* could not climb through trees as well as, say, chimpanzees. Chimpanzees have many adaptations in their arms and shoulders to let them hang from branches and climb vertically up trees with incredible speed. *Ardipithecus* lacked these adaptations, so it may have had to move less fluidly through the trees, using its hands and feet all at once to grip branches.

The closer we move to the present day, the richer the hominin fossil record becomes. Between 4 million and 2 million years ago, several species belonging to the genus *Australopithecus* appeared. Dart's small-brained

child belonged to a species known today as *Australopithecus africanus*. These australopithecines still had snouts and chimpanzee-sized brains. Weighing between 25 and 50 kilograms, they were about as heavy as a chimpanzee, too. They also still had long, curved toes and fingers that would have helped them to grasp branches. Their arms were relatively long, and their ankles could rotate more freely than ours.

On the other hand, australopithecines had even more adaptations for walking than *Ardipithecus ramidus* did. Their spines arched backward, so the upper body sat above the hips rather than extending forward. Their legs were straighter, and their knees were located beneath the midline of the body. Their feet bore many traits that are important for walking upright, such as a stout heel and the beginnings of an arch instead of a flat sole. Paleoanthropologists have even found hominin footprints from this period. A bed of volcanic ash that formed in Tanzania 3.6 million years ago preserved the marks of bipedal walkers—probably a species known as *Australopithecus afarensis* that lived in the area at the time (FIGURE 4.22). In 2009, scientists discovered a 1.5-million-year-old trackway in Kenya that preserved footprints nearly identical to those of living humans.



Kenneth Garrett/Getty Images.

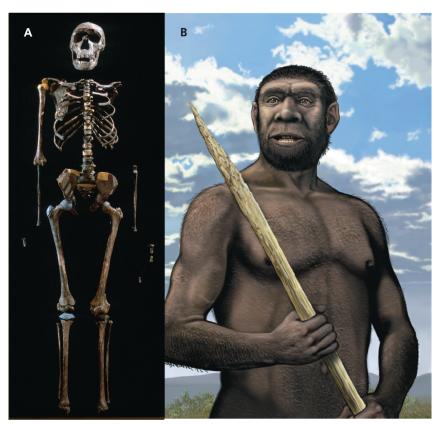
FIGURE 4.22

A volcanic eruption 3.6 million years ago in Tanzania preserved the footprints of bipedal hominins—probably *Australopithecus afarensis*.

Why did walking upright evolve in these hominins? A lot of evidence suggests that hominins were shifting to a new environment. Chimpanzees live mainly in dense forests, where they can pluck fruit from trees. By looking at the plant and animal fossils found alongside the early hominin fossils, paleontologists have determined that they were living in more open woodlands. In this new environment, hominins may have had to travel farther to find food. Upright walking is more efficient than walking on knuckles, and

so bipedal hominins could have saved energy on long walks (<u>Harcourt-Smith</u> and Aiello 2004).

As time passed, new lineages of hominins emerged. Some, such as *Paranthropus*, belonged to a separate branch of the hominin tree from our own. But others had traits that reveal their close kinship to us. *Homo erectus*, which emerged 1.9 million years ago, had a flatter face than earlier hominins and slender arms and legs that were well adapted for walking—and perhaps running—bipedally (FIGURE 4.23). The hominin brain was also evolving: *Homo erectus* had a brain ranging between 600 and 900 cubic centimeters, about twice as big as a chimpanzee's brain. (Our own brain is around 1400 cc.)



(A) Danita Delimont/Alamy. (B) Carl Buell.

FIGURE 4.23

A: About 2 million years ago, hominins evolved that were taller and had larger brains than their ancestors. B: Known as *Homo erectus*, this species spread out of Africa and across Asia.

This change in anatomy comes along with a new kind of fossil record: stone tools. The oldest stone tools known, which are about 2.5 million years old, were little more than small rocks with chipped edges. Over time, the tools become more sophisticated, including stones that hominins fashioned for chopping or crushing. Paleontologists found some of these tools near fossilized bones of wildebeests and other large grazing animals. They also found cut marks on the bones. Like forensic scientists at a crime scene, paleoanthropologists used this evidence to generate a hypothesis: the hominins were butchering the animals for their meat.

Hominins probably started out as scavengers, but at some point they began to hunt. Harvard paleoanthropologist Daniel Lieberman and University of Utah biologist Dennis Bramble have argued that early species of the genus *Homo* were already hunting, using their long legs and other new adaptations to run long distances to exhaust their prey (Lieberman and Bramble 2007). It's also possible that this new anatomy was the reason *Homo erectus* became the first hominin to spread out of Africa to Asia and Europe. *Homo erectus* was still alive in Indonesia as recently as 50,000 years ago. And on the Indonesian island of Flores, paleoanthropologists discovered the bones of tiny, small-brained hominins that lived as recently as 19,000 years ago. Some researchers argue they are a species that evolved from *Homo erectus* and have dubbed them *Homo floresiensis*. (Others believe they were *Homo sapiens*.) But *Homo erectus*, and potentially *Homo floresiensis*, were our distant cousins—not our ancestors.

Clues to the origins of our own species come from more human-like hominins that evolved in Africa and Asia several hundred thousand years ago. *Homo heidelbergensis*, for example, had a higher skull than earlier hominins and a jaw that no longer projected as far forward as that of *Homo erectus*. *Homo heidelbergensis* may have given rise to the Neanderthals in Europe and Asia about 300,000 years ago. Neanderthals were stocky and still had thick browridges, along with several other traits that set them off from humans, including bowed arm bones and a slight depression on the back of the skull. But along with these differences, they shared an impressive number of similarities with our own species. Their brains were as big as or bigger than human brains, and they used those big brains to make sophisticated stone tools and hunt big game.

In Africa, meanwhile, *Homo heidelbergensis* may also have given rise to our own species, *Homo sapiens*. The oldest fossils that share enough key traits with living humans to be considered part of our own species come from a site called Omo, in southwestern Ethiopia, that dates to about 200,000 years ago. Along with increasingly human-looking fossils, paleontologists are finding increasingly sophisticated tools from this period. By 270,000 years ago, African hominins were making finely crafted obsidian blades. But it was only after the emergence of *Homo sapiens* that our lineage began to leave behind symbols such as elaborate cave paintings. In <u>Chapter 14</u>, we'll return to the story of human evolution and consider how a mind capable of such creations came to be.

These four examples—tetrapods, mammals, birds, and humans—show how scientists use phylogeny to reconstruct evolutionary history. We can see how complex body plans emerge gradually from older ones. Evolutionary trees offer support for Darwin's argument that homology, in all its guises of adaptation, is the result of common ancestry. But like all insights in science, phylogenies also raise new questions of their own. What are the genetic changes that produced the tetrapod body plan, for example, or gave rise to new structures such as feathers? The answers, as we'll see in the next chapter, lie in the molecules that make heredity possible.

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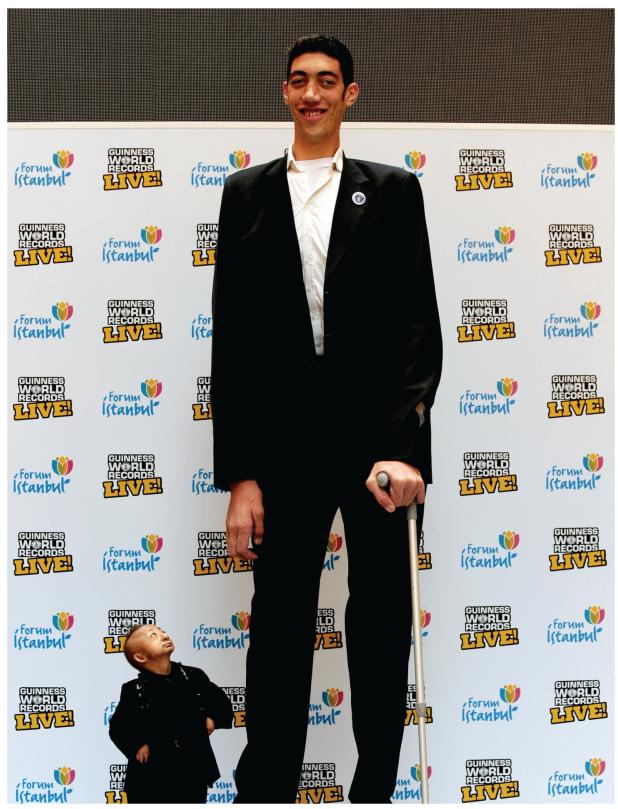
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MUSTAFA OZER/Getty Images.

The world's tallest man, 2.46-meter-tall Sultan Kösen, poses with shortest man in the world, He Pingping, standing at 74.61 cm. Kösen and He exemplify the striking range of human height—a trait that is influenced by both genetic variation and variation in environmental conditions.

Harvard geneticist Joel Hirschhorn studies how tall people are. Height might seem like the simplest thing a biologist could investigate. Hirschhorn doesn't need lasers to probe the inner structure of cells. He doesn't need high-speed video cameras to capture a thousand frames a second of a bat in flight. To measure someone's height, all Hirschhorn needs is a tape measure.

Yet the apparent simplicity of height hides a hidden world of complexity. Humans vary tremendously in the height they reach as adults. In any population of people, some individuals are very short and others are very tall. The average height varies from one population to another. In central Africa, Pygmies rarely reach more than 1.5 meters. In Denmark, men grow to an average height of 1.8 meters. The average height in many countries has gradually increased over the past century.



Joel Hirschhorn.

FIGURE 5.1

Joel Hirschhorn of Harvard University and his colleagues study DNA from tens of thousands of individuals to figure out why some people are tall and some are short.

Explaining all this variation is a staggeringly big challenge that has kept scientists busy for over a century. To address it, Hirschhorn collaborates with hundreds of scientists worldwide, studying tens of thousands of individuals. And yet they're still a long way from thoroughly explaining why we are as tall as we are.

Your height depends on many factors. The environment in which you grew up has a powerful influence. (The environment includes everything from the food you eat to the chemistry of your mother's uterus while she was pregnant with you.) Along with the environment, heredity also exerts an influence of its own. In an identical environment, short parents will tend to have short children, and tall parents will tend to have tall ones. To make things even more complex, the effect that the environment has on an individual

depends on his or her genes. Two people may respond to the same favorable environment by growing to different heights.

To untangle these factors, Hirschhorn searches for genes that help determine height. He and his colleagues examine the DNA of many people from around the world and look for variants of genes that show up more often in tall people or short ones. In a 2007 study of 4921 people, Hirschhorn and his colleagues discovered a gene, called *HMGA2*, that had a strong effect on height—the first time anyone had found such a gene (<u>Weedon et al. 2007</u>). People may have different versions of *HMGA2*, and having one version can increase a person's height by a centimeter.

Since then, Hirschhorn and his colleagues have cast a far wider net. In a study published in 2010, they analyzed data from 183,727 people (Allen et al. 2010). The huge number of subjects made it possible to find height-associated genes that had only small effects. As a result, in human DNA the researchers were able to identify 180 sites associated with height.

This fine-grained understanding of variation is far beyond what Charles Darwin possessed. He tried to understand it as best he could in the mid-1800s. He skinned rabbits and lined up their bones on his billiard table to measure their lengths. He spent hours in his hothouse studying orchids. In his study, he pored over a microscope to observe the anatomy of barnacles, noting the variations in size and shape within a single species.

Darwin knew enough about heredity and variation to make them cornerstones in his theory of evolution. But he never figured out how heredity worked. He came up with a theory that particles from the body streamed into eggs and sperm, blending together in offspring. But a Scottish engineer named Fleeming Jenkin pointed out a grave problem with this notion. Imagine that a bear had a thick coat better adapted to a cold climate than the coats of other bears. If it mated with a short-haired bear, their traits would be blended. In the next generation, the trait would become even more diluted. It was hard for Jenkin to fathom how natural selection could

drive traits to become more common if they were being blended away. Darwin didn't have a good response.

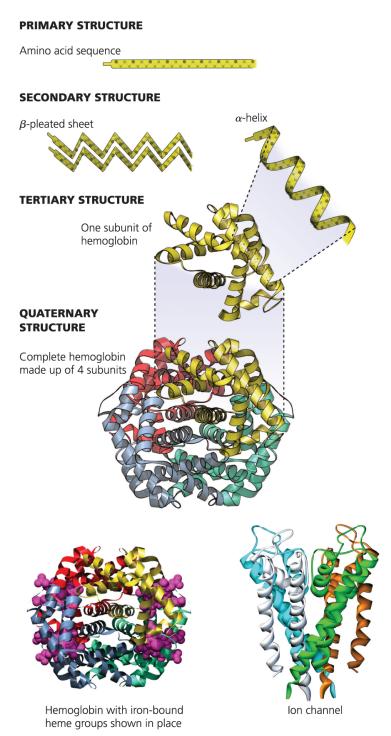
Incredibly, even as Darwin struggled with heredity, a Czech monk was uncovering the workings of genes. But Gregor Mendel's work would go ignored until 1900, eighteen years after Darwin's death. It was only in the twentieth century that the science of genetics was born, and biologists finally began to understand how evolution unfolds on the molecular scale.

Evolution's Molecules: Proteins, DNA, and RNA

The human body is made up of about a trillion cells, each of which is made up of millions of molecules. Three types are especially important: proteins, DNA, and RNA.

While water makes up most of the mass of a cell, proteins make up most of its dry weight. They not only give the cell much of its structure, but they also carry out many of the chemical reactions essential for life. Some proteins act as enzymes, breaking down molecules in the food we eat. Other proteins can store important molecules; red blood cells, for example, use the protein hemoglobin to bind oxygen so that they can deliver it from the lungs throughout the body. Proteins can also transmit information, relaying signals across a cell or even from one cell to another. After you eat a meal, for example, the level of glucose sugar in your blood rises, leading pancreas cells to secrete the protein insulin into the bloodstream. The insulin spreads through your body, signaling muscle cells to take up the extra glucose.

All told, the human body makes an estimated 100,000 kinds of proteins. Despite their astonishing variety, proteins are all made from the same small set of building blocks—a group of 20 molecules called amino acids. Cells assemble amino acids into long chains, and the charges of each amino acid attract it to some parts of the protein and repel it from others. These forces cause the protein to fold into a sheet, a cylinder, or some other complex shape. A group of proteins may come together to form an even more intricate structure. The final shape that proteins assume allows them to carry out their function (FIGURE 5.2).



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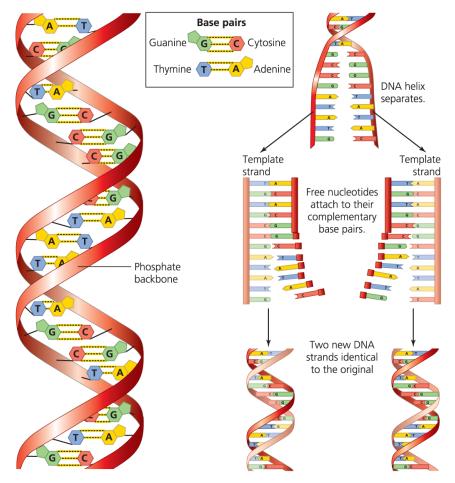
FIGURE 5.2

Proteins are chains of building blocks known as amino acids. They fold into complex structures and then join with other proteins to carry out sophisticated functions.

The sequence of amino acids in a protein is encoded in DNA. DNA is a long linear chain assembled from another set of building blocks, called nucleotides. Each nucleotide contains one of four bases, known as adenine, cytosine, guanine, and thymine (A, C, G, and T for short). You can think of these bases as the four-letter alphabet that spells out the genetic recipes for the ingredients of life.

The human genome—all the genetic information in a human cell—totals about 3.5 billion bases of DNA. When a cell divides, it creates a new copy of every one of those bases. It manages to perform this feat with almost perfect accuracy, thanks to its structure. A DNA molecule consists of two strings of nucleotides that wind together to form a double helix. Each base on a strand binds weakly to a base on the other strand. This pairing follows two simple yet powerful rules: adenine can bind only with thymine, and guanine can bind only with cytosine.

Thanks to these rules, the sequence of bases along one strand of DNA is perfectly matched to an inverse sequence on the other. If, for example, one strand of DNA contains the sequence ATTGTCG, we can be sure that its other strand will read TAACAGC. Although these two strands look different, they generally contain the same information. To make a new copy of its DNA, a cell pulls apart the two strands of the molecule (FIGURE 5.3). Each strand then serves as a template for the formation of a mirror-image strand. The result: two identical DNA double helixes.



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FIGURE 5.3

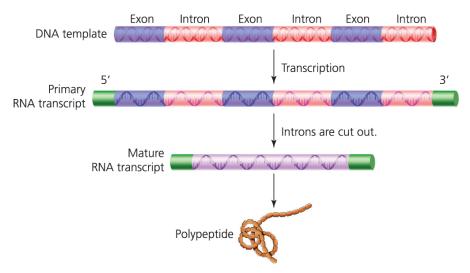
DNA forms a double helix. A cell replicates it by pulling apart the strands and assembling complementary ones.

Just about all living things use DNA as their genetic material, but they organize it in different ways. Humans and other eukaryotes keep their DNA coiled around proteins known as histones, so that they look like beads on a string. These coils are further arranged into tightly bundled rods known as chromosomes.

Humans have 46 chromosomes. Males and females have almost identical chromosomes, except for a single pair. In males, those two chromosomes are known as the X and Y chromosomes. The Y chromosome contains genes involved in determining the male sex. Females, on the other hand, have 23

identical pairs of chromosomes, including two X's. Other eukaryote species have different numbers of chromosomes. (Bacteria and archaea organize their DNA in a different way, which is presented in **BOX 5.2.**)

A cell takes an elaborate series of steps to turn the information encoded in DNA into proteins (FIGURE 5.4). It first has to unwind a segment of DNA that includes a region encoding a protein, known as a protein-coding gene. A swarm of proteins then converge on a spot near one end of the gene, called a promoter region. Once assembled there, they race their way from one end of the gene to the other. (An average human gene contains around 20,000 bases, although some genes have as many as 2.3 million bases.)



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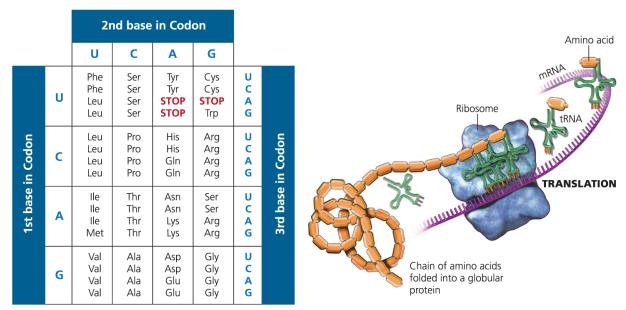
FIGURE 5.4

Proteins are encoded in genes. To produce a protein, eukaryotes transcribe a gene in the form of an RNA molecule. During this transcription, they edit out noncoding segments of the gene.

As the cluster of proteins travel along the gene, they assemble a new string of nucleotides, known as RNA. This process is known as transcription. The sequence of RNA corresponds to the original DNA sequence of the gene, but during transcription, the cell removes certain segments of the gene. The segments that stay in the RNA transcript are known as exons; the ones that get cut out are introns.

The cell can now use the RNA transcript to build a protein. Scientists call this process translation, because our cells have to translate the four-base system of nucleotides into the 20 different amino acids used to assemble proteins.

Translation takes place in a structure called the ribosome, a cluster of proteins and RNA molecules. It reads the RNA three nucleotides at a time and grabs a corresponding amino acid, which it attaches to a growing protein. The set of rules governing the translation of any particular trio of bases into specific amino acids is known as the genetic code (FIGURE 5.5).



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FIGURE 5.5

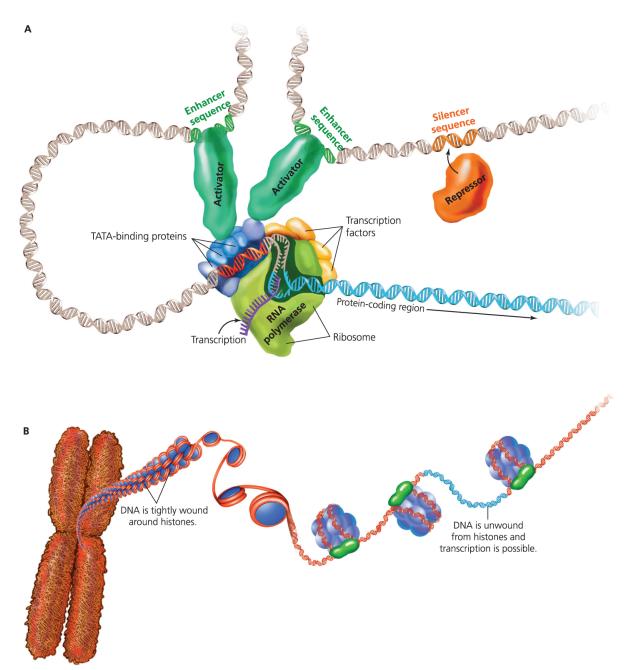
Once a messenger RNA (mRNA) molecule is transcribed from a gene, a ribosome uses it to assemble a protein. It reads three bases in the RNA at a time and then selects one of 20 amino acids. (Each type of amino acid is attached to a transfer RNA molecule [tRNA].) The chart shows the rules for this so-called genetic code.

Box 5.1

A Note on the Names of Genes and Proteins

Genes are typically italicized (such as *FOXP2*), while the proteins they encode have the same name but in roman type (FOXP2). *FOXP2* refers to the human version of the gene, *Foxp2* to the mouse version, and *FoxP2* in all other vertebrates that possess the gene. In some cases, the scientists who discovered and named genes did not recognize that the genes had already been found in other species. As a result, many orthologous genes have entirely different names. In <u>Chapter 8</u>, for example, we will learn how *Dpp* and *BMP4* are two versions of the same gene; *Dpp* is the version in invertebrates and *BMP4* is in vertebrates.

Humans have about 20,000 protein-coding genes. At any moment, each cell is expressing only a small fraction of those genes, and many of the genes expressed in one kind of cell are not expressed at all in another. The cells that produce our hair do not produce hemoglobin; likewise, our blood cells do not express the keratin proteins found in our hair. A complex network of molecules—including proteins and RNA molecules—regulates where and when genes are expressed (FIGURE 5.6). The expression of a gene can also be influenced by molecules that are produced by other cells. The hormone adrenalin, for example, is produced in the adrenal gland located on top of the kidneys. It circulates around the body and attaches to the surface of muscles and other types of cells, switching on other genes inside.



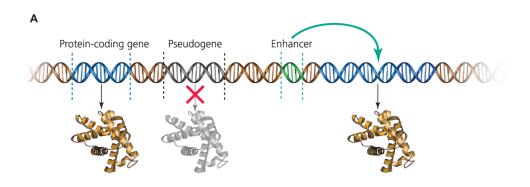
Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company A: Many proteins and RNA molecules cooperate to determine which genes get expressed in a particular cell in a particular moment. B: Eukaryote DNA is intricately wound into chromosomes. Proteins unwind portions of DNA to enable gene expression.

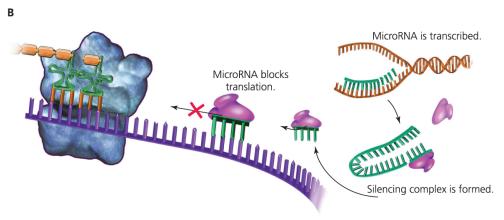
FIGURE 5.6

A: Many proteins and RNA molecules cooperate to determine which genes get expressed in a particular cell in a particular moment. B: Eukaryote DNA is intricately wound into chromosomes. Proteins unwind portions of DNA to enable gene expression.

The Hidden Genome

Protein-coding genes make up only about 1.2 percent of the human genome. The other 98.8 percent is a hodgepodge of elements, some essential for survival and some little more than baggage that gets carried from one generation to the next (FIGURE 5.7).





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FIGURE 5.7

A: Protein-coding genes make up only 1.2 percent of the human genome. The noncoding portion includes regulatory elements, pseudogenes, and genes encoding functional RNA molecules. B: Shown here is one type of RNA molecule, known as microRNA. This tiny piece of RNA can regulate the expression of other genes.

Much of this non-protein-coding DNA contains genes for RNA molecules. Instead of serving as a template for proteins, these RNA molecules carry out other functions. Some become parts of ribosomes. Some form caps at the ends of chromosomes. Many help regulate genes, switching them on and off in response to signals from outside the cell.

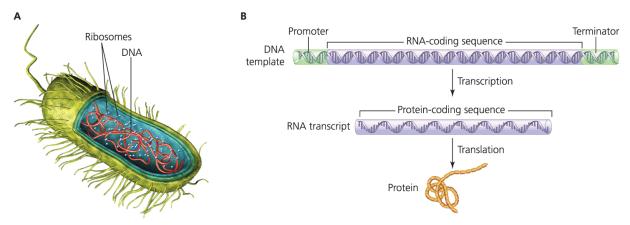
The human genome is also peppered with defunct genes. Originally they were functional, but mutations disabled them, turning them into pseudogenes ("fake genes"). In a recent study, scientists estimated that the human genome contains 17,032 pseudogenes (<u>Alexander et al. 2010</u>). In other words, there are almost as many pseudogenes in the human genome as there are proteincoding genes.

Over half of the human genome is composed of neither genes, nor vestiges of human genes, nor regions that regulate genes. Instead, it is made up of parasite-like segments of DNA, known as mobile genetic elements. They don't encode functional proteins or RNA molecules. Instead, they make new copies of themselves that are then inserted into another location in the genome. As we'll see in Chapter 12, many of these elements originated from viruses that inserted their genes into the DNA of our ancestors.

Box 5.2

Genes and Heredity in Bacteria and Archaea

Lukaryotes organize their DNA in a markedly different way than bacteria and archaea do (FIGURE 1). In bacteria and archaea, a single circular chromosome floats within the cell. It is not constrained by a nucleus, nor is it tightly spooled around histones. But this DNA is not simply a loose tangle. Bacteria and archaea produce proteins that keep sections of DNA organized in twisted loops. Like eukaryotes, bacteria and archaea regulate their genes with a network of proteins and RNA molecules, switching them on and off in response to their environment.

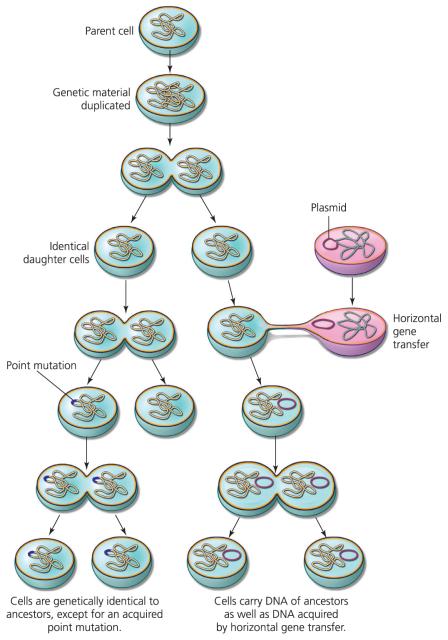


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FIGURE 1 A: The structure of cells in bacteria and archaea are different from that of cells in eukaryotes. The cells in bacteria and archaea lack a nucleus, for example, so their DNA spans much of their interior. While they have ribosomes, as do eukaryotes, they lack mitochondria and other organelles. B: Like eukaryotes, bacteria control gene expression with regulatory elements. However, transcription and translation are simpler in bacteria and archaea. They do not have introns and exons, for example. Consequently, the sequence in a gene corresponds much more closely to the corresponding RNA transcript.

In bacteria and archaea, individual cells typically grow until they are large enough to divide. They then build a second copy of their circular chromosome, after which the two DNA molecules are dragged to either end of the dividing cell. The two daughter cells are identical to the original, except for mutations that arise during DNA replication.

Bacteria and archaea have many of the same kinds of mutations found in eukaryotes, such as point mutations and insertions, which they pass down to their offspring. But mutations can also spread by other routes (FIGURE 2). Microbes sometimes contain additional DNA carried on small ringlets called plasmids. Under certain conditions, a microbe will translate some of the genes on a plasmid, producing proteins that form a tube, called a pilus. The pilus reaches out to another microbe, and once it makes contact, the donor cell can then pump a copy of the plasmid through it to the other cell. It often pumps a copy of some of its chromosomal DNA as well.



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FIGURE 2 Bacteria and archaea replicate by dividing in two. With each round of division, they make copies of their DNA. Except for mutations, the DNA of the descendants is identical to their ancestors'. Bacteria and archaea are more prone than eukaryotes to experience horizontal gene transfer as well. In this figure, the red microbe contains a ring of DNA called a plasmid. It forms a tube with the green microbe, through which the plasmid is transmitted. The recipient cell—and its descendants—now carry the donated DNA. Horizontal gene transfer is rare, but it's nevertheless a powerful factor in evolution—

for example, it drives the evolution of antibiotic resistance in populations of bacteria as well as between species.

In effect, plasmids are genetic parasites, using bacteria and archaea as their hosts and spreading to new hosts through the pili encoded in their own genes. But these parasites sometimes have benefits to their hosts; many plasmids carry genes that provide resistance to antibiotics, for example.

Horizontal gene transfer, as this movement of genes is called, does not depend solely on plasmids. Viruses that infect bacteria and archaea sometimes incorporate host genes into their own genome. When they infect a new host, they can insert those genes into their new host's chromosome. Meanwhile, some species of bacteria can slurp up naked DNA from the environment.

Horizontal gene transfer usually turns out to be a dead end. Foreign genes typically harm a microbe, slowing down its growth or killing it outright. When a microbe acquires a useful gene, on the other hand, natural selection can favor it. Evidence for successful horizontal gene transfer can be found in studies on the spread of antibiotic resistance: the same gene often turns up in different species. It's also possible to identify cases of horizontal gene transfer that occurred millions of years ago by performing large-scale comparisons of DNA in bacteria and archaea. In a number of cases, scientists have found genes in species that are unlike their close relatives but are homologous to genes found in distantly related clades. These studies indicate that horizontal gene transfer has been a major element of evolution. In *E. coli*, for example, 80 percent of all the genes in its genome show evidence of horizontal gene transfer at some point since the last common ancestor of bacteria (Dagan and Martin 2007).

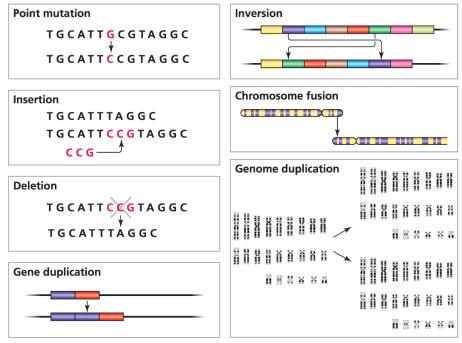
Mutations: Creating Variation

Cells copy DNA almost perfectly, but every now and then they introduce an error. These mistakes, known as mutations, are one of the main sources of variation. As a result, they make evolution possible.

Mutations can have many causes. Radioactive particles pass through our bodies every day, for example, and if one of these particles strikes a molecule of DNA, it can damage the molecule's structure. When the sun's ultraviolet rays strike our skin cells, they can cause mutations as well. Toxic chemicals also can interfere with DNA replication and trigger mutations. Even without these disruptions, mutations can still arise, because cells are not absolutely perfect in their replication of DNA.

Mutations can alter DNA in a number of different ways. Here are a few types of mutations (FIGURE 5.8):

- Point mutation A single base changes from one nucleotide to another (also known as a substitution).
- Insertion A segment of DNA is inserted into the middle of an existing sequence. The insertion may be as short as a single base or as long as thousands of bases (including entire genes).
- **Deletion** A segment of DNA may be deleted accidentally. A small portion of a gene may disappear, or an entire set of genes may be removed.
- **Duplication** A segment of DNA is copied a second time. A small duplication can produce an extra copy of a region inside a gene. Entire genes can be duplicated. In some cases, even an entire genome can be duplicated.
- Inversion A segment of DNA is flipped around and inserted backward into its original position.
- Chromosome fusion Two chromosomes are joined together as one.
- Aneuploidy Entire chromosomes are duplicated or lost.
- Genome duplication All the nuclear DNA in a cell is copied.



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FIGURE 5.8

DNA can mutate in several different ways.

The effects of mutations can vary enormously. Some have no effect. Some alter the shape of the protein encoded by a gene, changing its function. The mutant protein may carry out its original function at a faster or slower rate as a result. It may even start to take on a new function altogether. Nor does the size of a mutation necessarily determine the size of its effect on a trait. Large segments of DNA may be deleted without altering an organism's phenotype. Meanwhile, a mutation to a single nucleotide can cause a fatal genetic disorder or significantly increase an organism's chances of survival.

Even if a mutation doesn't alter the coding region of a gene, it can still have a noticeable effect. A mutation that changes the control region of a gene can change the number of proteins that are produced from it. It can cause a gene that's active in one organ to become active in a different one (FIGURE 5.9).





Mutar

(A) Dr. M.A. Ansary/Science Source. (B) Cartolano M., Castillo R., Efremova N., Kuckenberg M., Zethof J., Gerats T., Schwarz-Sommer Z., Vandenbussche M. (2007). A conserved microRNA module exerts homeotic control over Petunia hybrida and Antirrhinum majus floral organ identity. Nat. Genet. 39: 901–905.

FIGURE 5.9

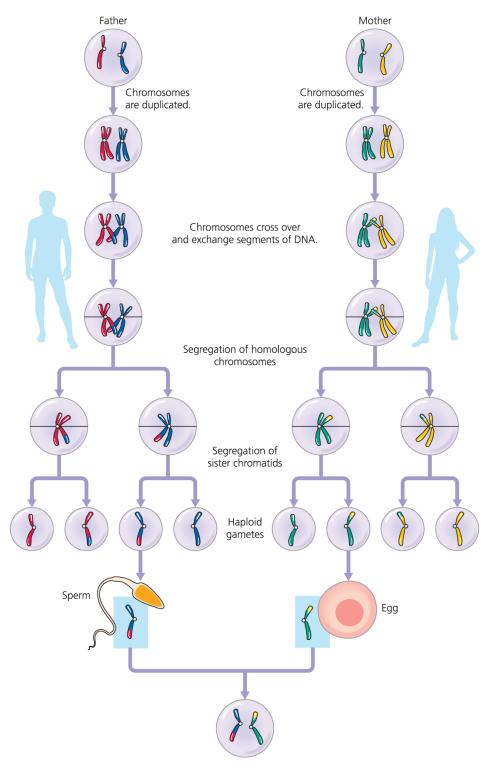
The effects of many mutations are subtle or even nonexistent. Still, even tiny mutations sometimes have large effects. A: Mutations as small as a single nucleotide variant can produce extra digits, such as the nine toes on this foot. B: A microRNA called MIRBL controls the identity of the parts of the petunia flower. A mutation to *MIRBL* causes the flower's petals to become stamens (<u>Cartolano et al. 2007</u>).

The rate at which mutations arise determines the variation on which evolution can act. In 2010 Leroy Hood, of the Institute for Systems Biology in Seattle, and his colleagues estimated the mutation rate in humans by sequencing the entire genomes of two human siblings and their parents and then searching the genomes of the children for mutations that their parents did not carry. All told, they identified 70 new mutations in each child (Roach et al. 2010).

Heredity

Once new mutations crop up, organisms pass them down to their offspring. Bacteria and archaea make a new copy of their genome, including any new mutations, and then divide into two cells (**BOX 5.2**). Things are more complicated in species like our own, which reproduce sexually and grow multicellular bodies. The cells in our brains, skin, heart, and other organs all grow and divide, and their DNA mutates along the way. But when we die, these cells die with us. Only eggs and sperm can carry their DNA into the next generation.

When a man and a woman have a family, the children are not identical clones, even though they have identical parents. That's because the children inherit different parts of their parents' DNA. The father's sperms and the mother's eggs carry only a single copy of each chromosome. Although each chromosome is nearly identical to its pair, it has many unique sequences. Which copy of each chromosome ends up in an egg or a sperm cell is usually just a matter of chance. And as this sorting happens, another source of variation emerges. The chromosomes cross over and exchange segments of DNA, in a process known as genetic recombination (FIGURE 5.10).



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Among sexually reproducing organisms, like humans, males and females combine their gametes to reproduce. Each gamete receives only one copy from each pair of chromosomes.

Every egg is thus genetically different from every other egg in a woman's ovaries; likewise, every sperm produce by a man is unique. As a result, siblings from the same parents are always different from one another, with the exception of identical twins.

Genetics in the Garden

The genetic makeup of an organism is known as its genotype. Its manifestation—the traits that the genotype produces, in other words—is known as its phenotype. Tracing the origin of the phenotype to the genotype is no easy task. A trait does not come with a label detailing all the genes that helped build it. Scientists must rely instead on indirect observations.

The first glimpse of how genotypes give rise to phenotypes was made by the Czech monk Gregor Mendel (1822–84) (FIGURE 5.11). Before entering a monastery, he had attended the University of Vienna and had become fascinated by heredity. After many years of reflection, he concluded that heredity was not a blending of traits, as many naturalists then believed. Instead, he believed that it came about by the combination of discrete factors from each parent.



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FIGURE 5.11

Gregor Mendel (1822–84) first recognized that inherited traits were made possible by factors (now called genes) passed down from parents to offspring.

To test his idea, Mendel planned out a long-term experiment. He grew a garden of pea plants of different varieties. Mendel settled on 22 different varieties and chose seven different traits to track. His peas were either round or wrinkled, for example. Their pods were yellow or green as well, and they were also either smooth or ridged. The plants themselves might be tall or short, and their flowers might blossom at their tips or along their stems.

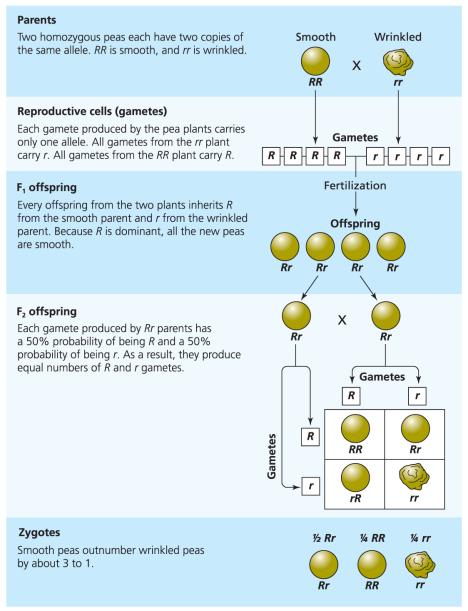
Delicately placing the pollen of one plant on another, Mendel created thousands of hybrids, which he then interbred. After crossing smooth and wrinkled peas, for example, he shucked the pods a few months later and found that the hybrid peas were all smooth. The wrinkled trait had utterly disappeared from sight. Mendel then bred these smooth hybrids together and grew a second generation. While most of the peas were smooth, some were wrinkled—just as deeply wrinkled as their wrinkled grandparents. The wrinkled trait had not been blended away as Fleeming Jenkin had argued. It had gone into hiding in the hybrids and then reappeared as strong as ever when the hybrids were interbred.

The number of peas that ended up wrinkled would vary on each plant, but, as Mendel counted up more and more of them, he ended up with a ratio of one wrinkled seed for every three smooth ones. He crossed varieties to follow the fate of other traits, and the same pattern emerged: one green plant for every three yellow ones, one white seed coat for every three gray ones, and one white flower for every three violet ones.

Mendel realized that he had found an underlying regularity to the confusion of heredity, which he described in a paper in 1865. But his contemporaries ignored his work, and he died at his monastery in 1884 with a reputation as having been little more than a charming putterer. But he was actually a pioneer in genetics, a field that didn't even come into formal existence until 16 years after his death.

After a hundred years of research, it is now clear why Mendel's peas grew the way they did (FIGURE 5.12). The difference between a smooth pea and a wrinkled pea is determined by a single gene that encodes an enzyme that helps break down sugar. This enzyme is called starch-branching enzyme (SBEI). Thanks to mutations, there were two versions of this gene in Mendel's peas. (Scientists call different versions of the same gene alleles.) The alleles of the gene for SBEI are now called *R* and *r*. Mendel's wrinkly

peas carried two copies of r, while the smooth peas either had RR, or an R from one parent and an r from the other.



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FIGURE 5.12

Gregor Mendel noticed that heterozygote peas produced offspring with a three-to-one ratio of certain traits. This figure shows how this ratio emerges as alleles for wrinkled or smooth peas are passed down through three generations. Some traits, such as the wrinkled texture of peas, are all or nothing. A pea is either wrinkled or it is not. This phenotype reflects the underlying genotype: a single gene influences it. The more genes

that influence a trait, the more possible genotypes exists, and thus more possible phenotypes.

Scientists refer to wrinkles on peas as a recessive trait, because two copies of the same allele are required to produce it. Smoothness is a dominant trait, because only a single *R* allele is enough to produce it. An organism that carries two copies of the same allele (*rr* or *RR* in this case) is called a homozygote. An organism with two different alleles of a gene is called a heterozygote.

The DNA sequence of the R and r alleles differs in one crucial way: the r allele contains 800 extra base pairs of repetitive DNA. This extra DNA prevents a pea cell from producing SBEI. Without SBEI, a pea can't break down sugar effectively, and so sugar levels go up. A sugary seed absorbs extra water as it develops, so that it swells to a larger size. Later, when the pea begins to dry out, it shrinks and its surface folds in on itself, forming wrinkles. (Bhattacharyya et al. 1990).

Things go differently in *RR* peas. They make the SBEI enzyme, so they can break down sugar and don't swell as much. When the *RR* peas dry, their smaller surface does not wrinkle, leaving them smooth. In heterozygotes, the *R* allele makes its normal supply of SBEI while the *r* allele makes none. Apparently, the SBEI produced from a single *R* allele is enough to keep peas from becoming wrinkled.

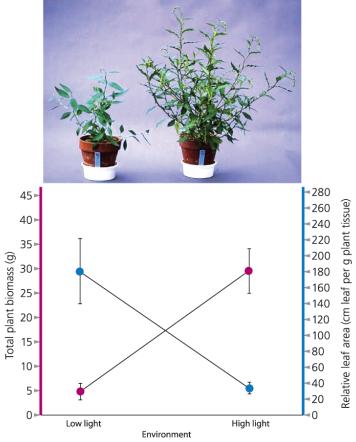
Now we can understand why Mendel discovered his three-to-one ratio of smooth and wrinkled peas produced from hybrids. Each Rr hybrid has a 50 percent chance of passing down either allele to its offspring. So a quarter of those offspring will be rr—wrinkly. The rest will be RR, Rr, or rR—in all cases, smooth. Looking at a single pea plant cannot tell you what probability it has of inheriting a particular allele, nor can it tell you the different effects of its R and r alleles. Only in a population—such as the population of peas in Mendel's garden—can these patterns emerge.

From Genotype to Phenotype: A Long and Winding Road

Mendel was able to decipher some of the rules of heredity because the phenotype he was studying came in relatively simple, discrete states. The peas were either wrinkled or smooth, for example, and not anything in between. And the variation in the phenotype was due entirely to the variation at a single place in the pea genome: the gene encoding SBEI.

There are other traits that have similarly simple links to their genotype. Huntington's disease causes certain kinds of neurons to waste away. Its victims slowly decline, losing the ability to speak and becoming unable to control their body movements. Geneticists have long known that Huntington's is a hereditary disorder that runs in families. In 1993, a team of American and British scientists finally pinpointed the cause of the disease: a mutation to a single gene, which produces a protein now known as huntingtin (Walker 2007). Ironically, scientists still don't know what the huntingtin protein actually does. But the gene's link to Huntington's disease is clear.

For most traits, however, the story is far less straightforward. They are the product of many genes, and the environment can produce variations that have no basis in DNA. Consider, for example, the plants shown in FIGURE 5.13. They were grown as genetically identical clones, but one was reared in low light and the other in abundant light. The low-light plant grew large leaves and small roots; the reverse was true for the plant in bright light. A single genotype thus produces two dramatically different phenotypes.



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Photo: Sonia E. Sultan/The Sultan Lab.

FIGURE 5.13

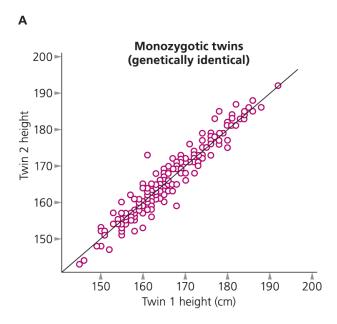
The environment strongly influences development. This photo shows two genetically identical plants that were reared in bright versus dim light, with their total mass (red circles) and relative leaf area (blue circles). The same genotypes produce dramatically different phenotypes in response to different environments. (Data from <u>Sultan 2000</u>).

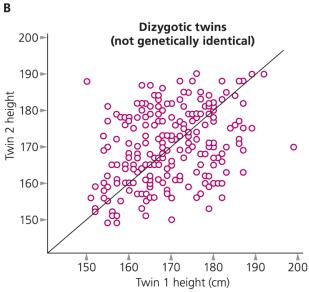
Scientists don't run these kinds of experiments on humans, of course, but sometimes history carries out unplanned experiments that reveal how the environment shapes our phenotype. In the 1970s Barry Bogin, an anthropologist who teaches at Loughborough University, began to study the short stature of the Maya people of Guatemala. Some scholars called them the Pygmies of Central America, because the men averaged only 1.6 meters tall and the women 1.4 meters. The other major ethnic group in Guatemala is

the Ladinos, who are descended from a mix of Mayan and Spanish ancestors. Ladinos are of average height.

The biggest factor in the difference between the Ladinos and the Mayas was not genes. It was poverty. The Mayas had less food and less access to modern medicine, which caused them to be shorter. During the Guatemalan civil war, which lasted from 1960 to 1996, a million refugees came to the United States. By 2000, Bogin found, American Mayas were 10 centimeters taller than Guatemalan Mayas, making them the same height as Guatemalan Ladinos. To grow much taller, the so-called Pygmies needed access to a decent diet for only a few generations (Bogin et al. 2002). They did not require new alleles.

At the same time, however, height shows a strong influence from genes. In 1903, for example, the British statistician Karl Pearson published data on 1100 families and showed that tall fathers tended to have tall children. Recently, David Duffy of the Queensland Institute of Medical Research and his colleagues surveyed twins (FIGURE 5.14; Duffy, personal communication). They compared identical twins (who share the same set of genes) to fraternal twins (who develop from separate eggs and thus share only some of the same alleles). When twins grew up in the same environment, identical twins ended up much closer in height than fraternal twins. This result strongly suggests that the differences between them arise from their genes.





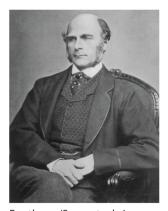
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FIGURE 5.14

A: This graph shows the relationship between the heights of identical twins. One twin's height is marked along the x-axis, and his or her twin sibling's height is marked on the y-axis. The tight correlation shows that they tend to be of similar height. B: Fraternal twins do not show as strong a tendency to be of similar height. The difference between these two results is due to the strong influence of genes on height. Identical twins inherit identical sets of genes, while fraternal twins develop from separate eggs. Because identical twins

share more genes, they have a stronger tendency to grow to the same height. (Data from David Duffy.)

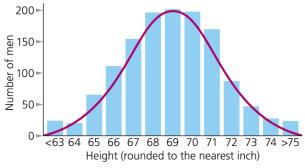
The contribution that genes make to variation in height is complex, however. Height, after all, is not an all-or-nothing trait like wrinkles on peas. Instead, there's a continuum in the human population. In 1885, Francis Galton (Charles Darwin's first cousin) made one of the first statistical studies of human height by gathering measurements of 1329 British men (FIGURE 5.15). When he plotted the distribution of heights on a graph, he found that height was distributed around a mean (FIGURE 5.16). Most of the men were of average or near-average height. Very tall and very short men were equally rare.



Pantheon/Superstock, Inc.

FIGURE 5.15

Francis Galton (1822–1911) studied the continuous variation in human height by collecting measurements from 1329 British men.



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FIGURE 5.16

Galton found that most men were close to average height, and the proportion of taller and shorter men was the same. Such bell-shaped curves are common in traits that are the result of many genes, each with a small effect. (Data from <u>Hartl 2007</u>.)

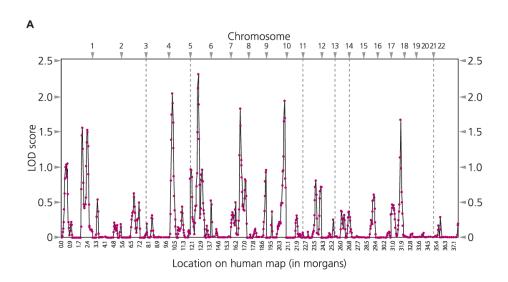
This kind of range in a trait is typically a sign that many genes are involved. Each gene influences the trait in the way Mendel originally hypothesized. But each gene exerts only a tiny influence. Because many genes are involved in the trait, people will have many different combinations of alleles. Most of the combinations will produce a trait close to average, and fewer will end up at the ends of the curve.

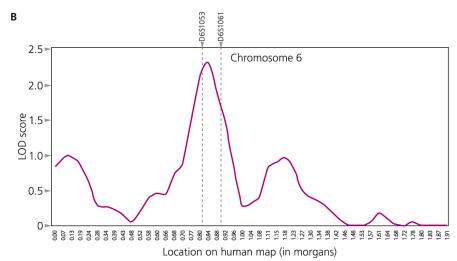
Searching for a lot of genes with small effects is much harder than looking for a few powerful ones. One way to look for them is to use a method called quantitative trait loci analysis (QTL analysis for short). Scientists search for genetic markers that tend to be found in people with high or low values of a particular trait.

This association may mean that a gene that influences a trait is located somewhere near the marker. Recall that in each generation, chromosomes swap segments. If two alleles are on different segments, they will become separated. If they are on the same segment, they'll get carried down to the next generation together. The closer two alleles are, the more likely they are to stay on the same segment of DNA during these swaps. Two people may both be tall because they have inherited the same allele from a common ancestor—along with a genetic marker nearby.

FIGURE 5.17 shows the result of one such survey. In 2004, Dutch researchers searched for QTLs associated with height in a group of 1036

people. They found that height was associated with a few genetic markers up to 100 times more often than you'd expect from chance (<u>Willemsen et al.</u> 2004).





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FIGURE 5.17

To find height-related genes, scientists look for genetic markers that tend to be found in people who are especially tall or short. This graph shows the result of one such survey, zooming in on one portion of chromosome 6. On the vertical axis, the LOD score stands for the "log of odds." An LOD of 2 means that a genetic marker is 100 times more likely to be associated with a height-related gene than not. Once scientists identify these regions, they can test nearby genes to see if they have an effect on height. The locations of two genes are marked here with dashed lines. (Data from Willemsen et al. 2004.)

Joel Hirschhorn, whom we met at the beginning of this chapter, and his colleagues have been expanding these searches for height genes to far larger groups of people. The more people they look at, the more sure they can be that the patterns they see are the results of real effects of genes, rather than random coincidences. They have now identified 180 candidate loci, including *HMGA2*, that can increase a person's height by a centimeter if he or she carries two copies of a particular allele (<u>Allen et al. 2010</u>). But simply identifying genes associated with a particular trait doesn't tell you how it helps produce that trait. Hirschhorn and his colleagues are just beginning to make those connections. Some genes influence height because they are expressed in the growth plates at the ends of the long bones in the arms and legs. There, they control the rate at which new cartilage forms (<u>Liu et al.</u> 2012).

It's a tremendous accomplishment to identify the first genes for height, but it's also important to put this discovery in perspective. All of the height-influencing genes identified so far account for just 10 percent of the phenotypic variation of height. Over a century after Galton began to explore height, scientists still have hundreds more genes to discover.

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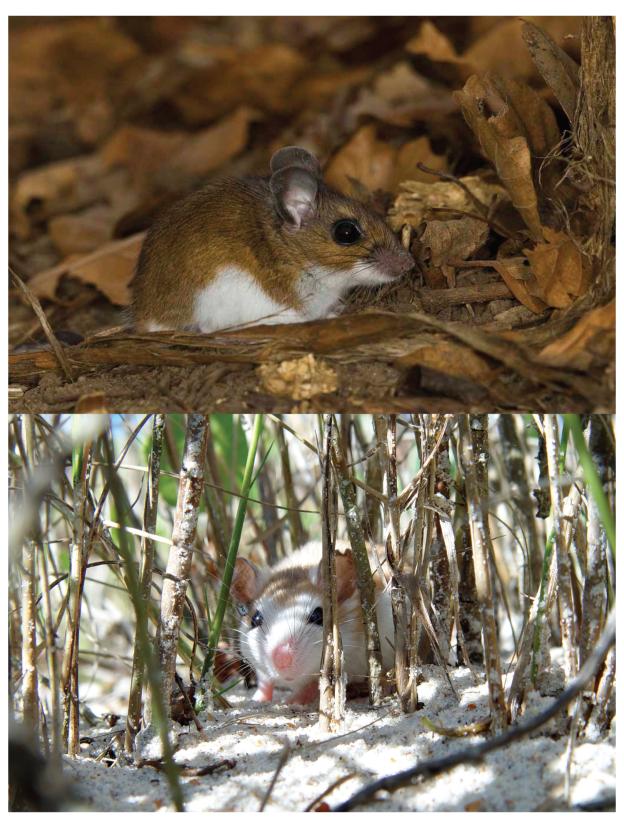
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The Ways of Change

Drift and Selection



Top photo: Shawn P. Carey/Migration Productions; Bottom photo: J.B. Miller/USFWS.

Oldfield mice that live in fields and forests have dark coats (*top*). Populations that have colonized beaches have become mostly white (*bottom*). Evolutionary biologists have pinpointed some of the mutations that natural selection has favored to produce this change.

Each February, Hopi Hoekstra does what many of her fellow New Englanders do: she gets on a plane and flies south to spend a couple of weeks on a Florida beach. But for Hoekstra, a biologist at Harvard, the journey is not a vacation. On the beaches of Florida, she and her colleagues can uncover the molecular basis of natural selection. It's a privilege Darwin could only dream of.

Walking along the snow-white dunes, Hoekstra and her students lay small metal boxes in the sand, each baited with food. Later, they return to find small visitors trapped in some of them: oldfield mice (*Peromyscus polionotus*). The mice live in tunnels under the sand dunes, coming out at night to gather seeds. They typically have white bellies and flanks, along with a narrow band of light tan down their backs (<u>Hoekstra 2010</u>).



From, "Melding Mannals and Molecules to Track Evolution" by Elizabeth Pennisi. Science 325:1332 (2009). Reprinted with permission from AAAS. Photo by Lynn Johnson/ National Geographic Society.

FIGURE 6.1

Hopi Hoekstra, an evolutionary biologist at Harvard University, has pioneered the study of the evolution of oldfield mice.

Oldfield mice are not limited to living on beaches. They also live inland, digging tunnels in the dark loamy soil of abandoned farm fields and open woodlands. Unlike the pale beach mice, the mainlanders have coats that are dark brown on top, with just a small patch of white on their underside.

Each kind of mouse is exquisitely well matched to its environment. The light coat of the beach mice blends into the white background of the dunes, while the dark coat of the inland mice resembles the dark soil over which they scamper. The mice do not develop their color in response to the habitat where they grow up; the colors are instead encoded in their genes. When scientists breed oldfield mice in captivity, parents with dark coats will produce mostly dark offspring; light-coated mice tend to produce light-coated pups.

And yet these genetic differences must have evolved recently. The Florida beaches and barrier islands that are home to the beach mice emerged from the ocean only 4000 to 6000 years ago. Dark mainland mice colonized these new habitats and evolved a new coat color in a matter of millennia.

Darwin and Wallace laid out their arguments for evolution long before scientists discovered that DNA makes heredity possible. After scientists established the science of genetics in the early 1900s, they turned their attention to evolution, merging the two sciences together. In this chapter, we'll see how that union has helped to give scientists a precise understanding of natural selection and other mechanisms of evolution. Armed with this understanding, Hoekstra and other scientists are starting to document evolution unfolding around us over the course of millennia, years, and even days.

The Rise and Fall of Mutations

As we saw in the last chapter, new mutations arise in every generation. If the organisms that carry them reproduce, those mutations can be transmitted to the next generation, and the next one after that. Over time, the mutations may become more and more widespread. Or they may dwindle away until they vanish.

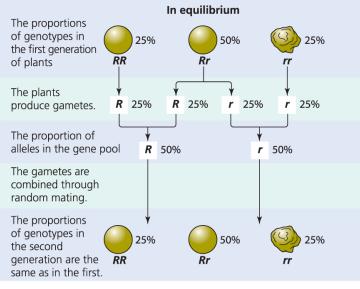
Mutated genes are a bit like newfangled words. Every year, for example, many new English words emerge in speech and in writing, but only a few of them prove successful, sweeping across the country and ultimately ending up in the dictionary. They may endure for centuries, long after the people who invented them have died. But most new words fail to take. Some of them may linger on in small circles, whose members use them to describe obscure things. Many new words survive only for a few years before sinking into oblivion.

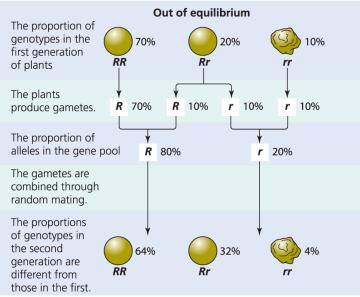
Change over Time—or Not

Evolutionary biologists can gain insights about the rise and fall of mutations by using simple mathematical models. One of the most important of these insights came in the early 1900s: Under certain conditions, the frequency of alleles will not change from one generation to the next—at least as long as those conditions hold. In other words, the population does not evolve.

This persistence is known today as Hardy–Weinberg equilibrium. It is named after the British mathematician G. H. Hardy and the German physiologist Wilhelm Weinberg, both of whom independently discovered it in 1908. FIGURE 6.2 illustrates the Hardy–Weinberg equilibrium in two populations of peas. In the first population, 25 percent of the peas are rr, 50 percent Rr, and 25 percent RR. Every pea produces gametes, each of which receives only one allele. There's a 50 percent chance of the allele in a gamete being either copy carried by the plant. Half of the gametes produced by the heterozygotes will carry R, and the other half will carry r. Since the homozygotes have two copies of the same allele, the RR plants will produce only R gametes, and the rr plants will produce only r gametes.

The Hardy-Weinberg Model





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FIGURE 6.2

The Hardy–Weinberg model shows how the frequency of alleles can change from one generation to the next. Top: In the first generation, 25 percent of the peas are wrinkled (rr homozygotes). Half of the peas are heterozygotes (Rr), and 25 percent are RR homozygotes. Each pea produces gametes. For simplicity, the model assumes that the alleles in a pea's gametes are proportional to the alleles in the pea. (For example, half of the gametes produced by Rr heterozygotes are R, and the other half are r.) As the third row shows, half of all the gametes produced by all the peas are R and the other half are r.

If we assume that these gametes all combine randomly with other gametes, half of the peas will inherit R and half will inherit r. As a result, the second generation will have exactly the same frequency of genotypes as the first. Bottom: In this case, the proportions of genotypes are different, with many more RR homozygotes, and fewer Rr heterozygotes and rr homozygotes. If the Hardy–Weinberg model begins with these peas, we end up with a different frequency of genotypes—fewer homozygotes and more heterozygotes. If this model is extended to the third generation, the proportions will be the same as in the second. Biologists call this unchanging condition Hardy–Weinberg equilibrium.

If we tally all the gametes produced by all the peas—*RR*, *rr*, and heterozygotes—50 percent will carry *R*, and the other 50 percent will carry *r*. To produce the next generation, the gametes combine randomly to produce new pea plants, each of which now carries two alleles, randomly selected from the pool of gametes. There's a 50 percent chance that each will be *R* or *r*. This means that the next generation will be 25 percent *rr*, 25 percent *RR*, and 50 percent *Rr*.

In other words, the peas pass on the same proportion of alleles to the next generation—and the frequency will stay the same in the next generation, and every generation into the future. Since *r* is recessive, 75 percent of the peas will be smooth and 25 percent will be wrinkly—the same proportions as before. This population of peas is, in other words, in Hardy–Weinberg equilibrium.

But what if you start off with a different mix of peas in your garden? Imagine that only 10 percent of the peas carry two copies of r. Only 20 percent are heterozygotes (Rr), and 70 percent carry two copies of R. Only 20 percent of the gametes produced by this population will carry r, and 80 percent will carry R. It turns out that, in the next generation, only 4 percent are rr, 32 percent are Rr, and 64 percent are RR. The new generation is different from the previous one. The proportion of wrinkly peas has dropped to less than half its original level.

In the following generation, something quite surprising happens: nothing. The proportion of gametes in the third generation will be 80 percent R and 20 percent R; just as it was in the second generation. And the plants they give rise to will be 4 percent R, 32 percent R, and 64 percent R—exactly the

same proportions as in the previous generation. The same will hold true of the fourth generation, and all the ones that follow, as long as the conditions of the model remain the same. This population has also reached Hardy—Weinberg equilibrium.

All mathematical models are only as good as the assumptions they are based on. For their models, Hardy and Weinberg assumed a population was infinitely large. While no real population meets this requirement, very big ones behave pretty much like the model. Another requirement for Hardy—Weinberg equilibrium is that every allele at the locus in question must be equally likely to be passed down to the next generation. In other words, no alleles can be lethal, nor can they give individuals a reproductive boost. Nor can any organisms enter or leave the population, because they could change the frequency of the alleles. Nor can new alleles enter the population.

All these requirements might seem to make the Hardy–Weinberg equilibrium far too abstract to be of any use in understanding evolution. And yet it turns out to be a powerful tool. Scientists can look at the frequencies of alleles of a locus in a real population and determine if they're in Hardy–Weinberg equilibrium. If they're not, then the scientists know that something is pushing the population away from equilibrium. They can then investigate hypotheses about what that factor is—and thus uncover evolution at work.

Here's an example of how scientists can use the Hardy–Weinberg equilibrium in the real world (FIGURE 6.3). In the 1970s, Luigi Luca Cavalli-Sforza of Stanford University and his colleagues studied variations in the genes that encode hemoglobin, the molecule that transports oxygen through the bloodstream (Stone, Lurquin, and Cavalli-Sforza 2007). They surveyed 12,387 adults in Nigeria, noting whether they had either of two alleles at a locus encoding part of the hemoglobin molecule called β -globin. They named these alleles A and S.

Genotype	Expected by HW	Observed
AA	9527.2 (76.9%)	9365 (75.6%)
AS	2672.4 (21.6%)	2993 (24.2%)
SS	187.4 (1.5%)	29 (0.2%)
Total	12,387	12,387

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FIGURE 6.3

Geneticists measured the frequencies of two alleles, known as *A* and *S*, of a gene that encodes part of hemoglobin. They then determined the frequencies of homozygotes and heterozygotes that would be expected if the alleles were in Hardy–Weinberg equilibrium. As this chart shows, the frequencies are significantly different from the expected values. As we'll see later, natural selection is responsible for the difference between these expectations and the actual frequencies. (Data from <u>Cavalli-Sforza 1977</u>.)

Cavalli-Sforza and his colleagues found that 87.7 percent of the alleles in the Nigerian population were *A*, and 12.3 percent were *S*. If these alleles were at Hardy–Weinberg equilibrium, then we'd expect that the percentage of people with the *AA* genotype would be 87.7 percent times 87.7 percent, or 76.9 percent. Likewise, only 1.5 percent of the population should be *SS*, and 21.6 percent should be *AS* heterozygotes.

When Cavalli-Sforza and his colleagues looked at the actual population of Nigerians, however, that's not what they found. The number of people with the SS genotype was more than seven times lower than predicted by the Hardy–Weinberg equilibrium. The number of people with the AA genotype was slightly lower than predicted, while the AS heterozygote was more common. In other words, the reality the scientists found in Nigeria violated the requirements of the Hardy–Weinberg model. That means they must be experiencing some sort of evolution.

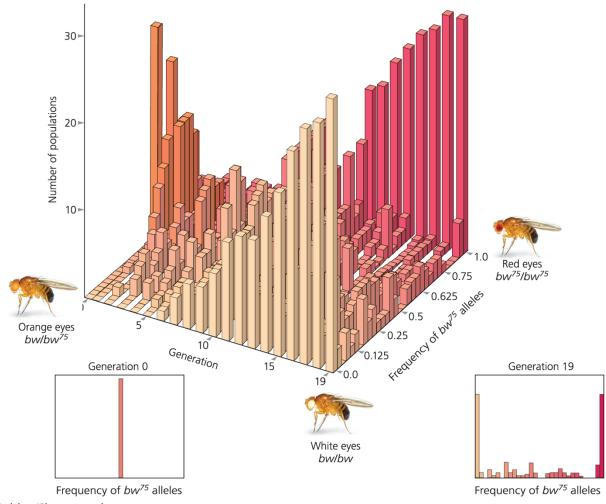
What sort of evolution might that be? Several different potential mechanisms of evolution may be at work in a case like this one. In this chapter, we'll take a close look at two of the most important of them: genetic drift and natural selection.

Genetic Drift: Evolution by Chance

In the 1950s, Peter Buri, a biologist at Iowa State University, bred thousands of orange-eyed flies (Buri 1956). The flies, known as *Drosophila melanogaster*, were heterozygous for a gene called bw^{75} that influences the color of their eyes. Each of Buri's flies carried an allele called bw and another called bw^{75} . It's very easy to tell a bw/bw^{75} heterozygote from homozygotes. A fly with two copies of bw has white eyes, while the eyes of a fly with two copies of bw^{75} are bright red. The heterozygote flies, on the other hand, have orange eyes.

Buri put heterozygous flies into 107 flasks. Each flask contained eight males and eight females. The flies mated and produced a new generation of flies. From each batch, Buri randomly chose eight males and eight females to breed again. For 19 generations, Buri bred the flies this way, keeping track of the colors of their eyes.

If the flies were in Hardy–Weinberg equilibrium, the frequency of the alleles would have remained fifty-fifty throughout the entire experiment. But, as **FIGURE 6.4** shows, they most certainly did not. The bw allele became rarer and rarer in some populations until it disappeared completely, leaving only bright red-eyed bw^{75}/bw^{75} flies. (Scientists refer to an allele that reaches 100 percent frequency as being "fixed.") In some of Buri's other populations, bw^{75} disappeared and bw became fixed. The remaining populations ended up falling between these two extremes.



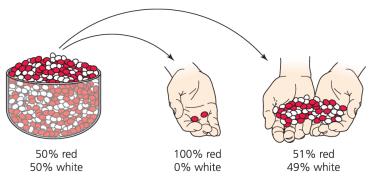
Roblan/Shutterstock.

FIGURE 6.4

This graph charts a series of experiments in which 107 populations of 16 fruit flies apiece were allowed to reproduce for 19 generations. At the end of the experiments, the *bw* allele had either become fixed or disappeared from many populations. (Data from <u>Hartl 2007</u>.)

Buri's flies violated only one requirement for Hardy–Weinberg equilibrium, but they violated it in a big way. Far from being infinite in size, their populations were extremely small. And in small populations, alleles can become fixed thanks only to chance. The evolution that Buri witnessed is known as genetic drift. The name comes from the way allele frequencies "drift" randomly away from their starting value.

To understand how drift happens, imagine yourself at a party. Your host has put out a big bowl of jelly beans that are half red and half white. Without looking into the bowl, you grab a few (FIGURE 6.5). If you pick out only two jelly beans, you would not be surprised to find that both are red. Even if you were to take four jelly beans, it wouldn't be a shock to find that they were not half white and half red. But the bigger your handful of jelly beans, the higher the odds become that you'll ended up with something close to a fifty-fifty split between the two colors. In other words, your handful of jelly beans resembles the bowl that they came from.



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FIGURE 6.5

To understand genetic drift, imagine alleles in a population as jelly beans in a bowl. If you only select a few jelly beans, you have a good chance of ending up with a different proportion of colors in your hand than in the bowl. If you grab a large handful, you're more likely to end up with similar proportions. In small populations, alleles may become more or less common from one generation to the next through a similar random process.

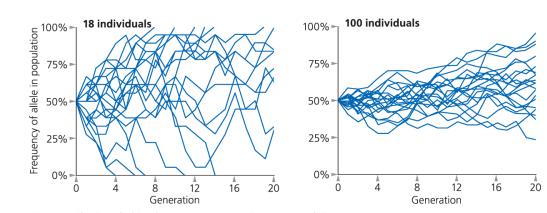
In Buri's experiment, the fly alleles behave like the jelly beans in a bowl, statistically speaking. The first generation of flies made a vast number of eggs and sperm, half of them carrying bw, and the other half carrying bw^{75} . If those gametes were mixed together randomly to produce many new flies, it would be like taking a large handful of jelly beans. In other words, chances would be very good that the alleles in next generation would be close to fifty percent bw and fifty percent bw^{75} alleles, just like the previous one.

But Buri bred only a few flies, not a lot. By taking only 32 gametes from each generation to produce the next one, he was, in effect, taking a few jelly

beans from the genetic party bowl. As a result, the odds were much more likely that he would end up with something other than a fifty-fifty split between the alleles.

These strokes of luck meant that in some of Buri's experimental populations, bw became rarer. In some of those populations, bw became rarer and rarer until it disappeared. A single fly was left with the allele, and when it reproduced, it passed down bw^{75} instead of bw. The same kind of fluke left other populations without any copies of bw^{75} .

The smaller a population, the easier it is for random drift to drive alleles to fixation. We can see this effect by looking at a computer simulation of populations at different sizes (FIGURE 6.6). In each generation of the simulation, a number of individuals are randomly picked to reproduce, forming the next generation of the population. All three simulations started off with an allele at a frequency of 50 percent. In small populations, drift causes alleles to become rapidly fixed or lost. In the largest populations, genetic drift is far weaker: by the end of the simulation, the frequency of the allele is still close to 50 percent.



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FIGURE 6.6

Left: This graph shows the results of several computer simulations of a population of 18 individuals. They tend to experience strong genetic drift, as an allele either becomes fixed or disappears. Right: In simulations with 100 individuals, genetic drift is weaker. Each new generation is much more likely to have the same frequency of the allele as the previous one. (Data from <u>Hartl 2007</u>.)

Bottlenecks and Founder Effects

Northern elephant seals (*Mirounga angustirostris*) crowd many beaches along the west coast of North America, where they haul themselves out of the water to mate and rear pups. The population of northern elephant seals in California alone is a prosperous 124,000. In the early 1990s, a biologist named Rus Hoelzel, then at the University of Cambridge, traveled to California to study the seals' genetic diversity.

He and his colleagues gathered tissue samples from two populations of northern elephant seals and extracted their DNA (<u>Hoelzel et al. 1993</u>). The scientists sequenced a 300-base stretch of DNA that has an especially high mutation rate. Mammal populations typically have 30 different alleles at this particular locus. But when Hoelzel and his colleagues looked at the locus in the elephant seals, they could find only two alleles.

The history of elephant seals holds the secret to their low genetic diversity. In the 1800s, hunters slaughtered them in huge numbers, and by turn of the century they were nearly impossible to find. Scientists estimate that they may have dropped to as few as 30 individuals. In such a tiny population, genetic drift became very strong, eliminating many alleles. To understand how this occurs, think back to the bowl of jelly beans in Figure 6.5. In that example, there were only two colors of jelly beans in the bowl. Imagine an entire barrel of thousands of jelly beans in 100 different colors, some of them common and some of them rare. If you scooped out 1000 jelly beans, you'd get most or all of those colors. But if you grabbed only 10, you'd lose most of the variation in the barrel. In a rapidly shrinking population, alleles disappear in a similar way, leaving a much lower level of genetic diversity.

But the elephant seals avoided extermination. Hunters stopped killing them, and by 1922, the population had grown to 350 animals. They came under government protection and have been recovering ever since. Their population has increased far faster than the rate at which new mutations have arisen. As a result, their genetic diversity remains low, over a century since they were nearly driven to oblivion. Scientists refer to the loss of genetic

diversity through a rapid reduction in population as a genetic bottleneck (FIGURE 6.7).



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FIGURE 6.7

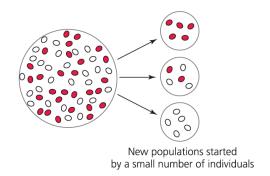
Northern elephant seals experienced a population bottleneck in the 1800s when their numbers shrank to about 30. They lost much of their genetic diversity, which has not increased much as their population has expanded.

The history of northern elephant seals stands in stark contrast to their southern cousins. Southern elephant seals never faced extinction, because they rear their pups on isolated islands where they aren't quite so easy for hunters to kill. The population of southern elephant seals probably never dipped below 1000 before hunting was banned. And that difference is reflected in their genetic variation. While Hoelzel found only two alleles in northern elephant seals, he found 23 in southern elephant seals (Hoelzel 1999).

A special kind of bottleneck, called the founder effect, occurs when a small number of individuals leave a larger population and colonize a new habitat. Seeds, for example, sometimes stick to the feet of migratory birds, which may carry them thousands of miles away to new homes. Because only a few seeds at most founded these new populations, they have a low genetic diversity (Popp, Mirré, and Brochmann 2011). The same holds true for human populations.

In 1789, for example, a few mutineers from the British ship HMS *Bounty* settled on Pitcairn Island in the middle of the Pacific Ocean (FIGURE 6.8). Twenty-seven adults and one baby founded the new population, and together they and their descendants lived in almost complete isolation for decades. By 1856 they were no longer able to support themselves on Pitcairn, so 193 residents moved to Norfolk Island. Today, Norfolk supports a population of 2000, most of whom are descended from the original founders of Pitcairn. But their genetic diversity remains low compared to populations of equal size that didn't go through such a drastic bottleneck (Macgregor et al. 2010).





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FIGURE 6.8

Left: Mutineers from the ship HMS *Bounty* on April 29, 1789. A party of 28 crew members and Polynesians settled the tiny and remote Pitcairn Island, and for most of two centuries, this island and later Norfolk Island were populated by descendants of this founder population. Right: Founder populations such as the one on Pitcairn Island have low genetic variation, because they don't capture the full diversity of their source population.

As we'll see in <u>Chapter 15</u>, founder effects can lead to unusual medical problems in isolated populations. On Norfolk Island, for example, a quarter of the people have a hereditary form of migraine headaches, a rate far higher than in large populations (<u>Maher et al. 2012</u>).

Selection: Winning and Losing

Genetic drift is not the only mechanism that can change the frequency of alleles in a population. Another is selection. Darwin and Wallace both proposed that selection could drive evolution, because some organisms reproduced more successfully than others. Their descendants then inherited their traits, including the ones that made them more successful. Darwin and Wallace came up with their concept of fitness long before biologists understood how traits are encoded by genes. Today, evolutionary biologists think about selection in terms of the success that different genotypes have in getting replicated. They call this success fitness.

To understand the concept of fitness, bacteria are a good place to start, because they have a simpler style of replicating their genotypes. Imagine a species of bacteria thriving in a pond, where many genotypes are found in the water. One way to measure the fitness of these strains is to compare their growth rates. We can give the genotype with the highest fitness a value of 1, and measure the relative fitness of the other ones as a fraction of it. If a slow-growing strain of bacteria in the pond grows at 80 percent of the rate of the fastest one, for example, its relative fitness is 0.8.

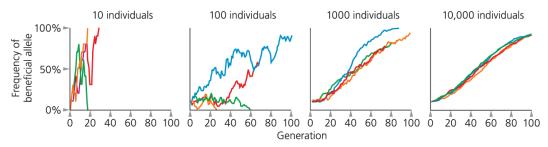
As new mutations arise in the pond, they may increase the fitness of a genotype, lower it, or have no effect. When an allele lowers the relative fitness of a genotype, it experiences negative selection. The most extreme case of negative selection a pond bacterium could experience would be a lethal mutation. With no descendants at all, its fitness would drop to zero. But mutations can still experience negative selection if they're not lethal. All they have to do is lower the rate of growth. A perfectly healthy microbe might have low fitness if it is slower to divide than other microbes. Over the course of many generations, the fraction of the population carrying that genotype will shrink. If they continue to undergo negative selection, these organisms will disappear completely.

Positive selection takes place when an allele increases the relative fitness of a genotype. Imagine that someone dumps a barrel of toxic chemicals into the pond full of bacteria. Most of the bacteria die or barely survive. But

some carry a mutation that lets them keep on growing, albeit slowly. Even at this slow rate of growth, the resistant genotype has the highest fitness and will become more common. Now imagine that the chemical settles down into the mud at the bottom of the pond, and the water recovers. The resistant genotype no longer has an advantage. In fact, another genotype may be able to grow faster under the new conditions.

Even a small difference in relative fitness can have big long-term effects on a population. That's because populations grow like investments earning interest. If you invest \$100 in a fund that earns 5 percent interest each year, your fund will increase by only \$5 in the first year. But in the second, it will increase by \$5.25. In every subsequent year, the fund will increase by a larger and larger amount. In 50 years, you'll have more than \$1,146. Because of this accelerating growth, even a slightly larger interest rate can lead to much bigger returns in the long term. If your bank raises your interest rate from 5 percent to 7 percent, you'll make only an extra \$2 in the first year. In 50 years, however, the fund will be more than \$2,945—close to triple what an interest rate of 5 percent would yield. Growing populations work much the same way. There may not seem to be a big difference between a genotype with a fitness of 1 and another of 0.95. But over time, such small differences can allow an allele to become fixed in a population.

The compounding power of natural selection is strongest in a large population and weakest in a small one. In a small population, it is opposed by strong genetic drift, which can eliminate beneficial mutations. In large populations, genetic drift is weak, allowing beneficial mutations to gain a foothold. FIGURE 6.9 shows the results of a computer simulation in which an allele with a selective advantage of 5 percent is added to populations of different sizes. In the big population of 10,000 individuals, it invariably becomes more common. In a population of 10 individuals, on the other hand, it disappears from half of the simulations. High relative fitness, in other words, is not a guarantee that an allele will spread—or even persist—in a population, because the effects of drift can be stronger than those of selection when populations are very small.



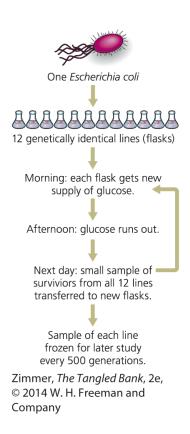
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FIGURE 6.9

Natural selection becomes more effective at higher population sizes. These graphs show the results of computer simulations of a population in which an allele that raises fitness by 5 percent is added to populations of different sizes (each colored line represents a different simulation). In all cases, the allele starts out with a frequency of 10 percent. The subsequent changes in its frequency result from the combined action of selection and drift. In the smallest populations, the allele disappears from half the simulations, even though it has beneficial effects on fitness. But in large populations, the allele becomes more common in all of them. (Data from Bell 2008.)

Fifty Thousand Generations of Selection: Experimental Evolution

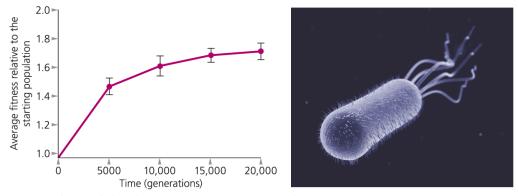
Fitness is not some abstraction found only in mathematical models. Scientists can measure it precisely in the real world. Some of the best measurements have taken place in the laboratory of Richard Lenski at Michigan State University (Barrick et al. 2009). Lenski started the experiment in 1988 with a single *E. coli* bacterium. He allowed the microbe to produce a small group of genetically identical descendants, and from these clones, he started 12 genetically identical populations of bacteria (FIGURE 6.10). Each population grows in 10 milliliters (ml) of broth that contains an assortment of nutrients the bacteria need to stay alive, including a scant amount of glucose.



Richard Lenski and his colleagues have used this method of breeding bacteria for over 20 years.

Once Lenski established the 12 identical lines, one of his students or technicians would draw out 0.1 ml from each flask and drop it into a new flask full of fresh glucose. Under these conditions, the bacteria doubled their population on average 6.67 times a day. From time to time, they occasionally acquired new mutations. Alleles that lowered their fitness experienced negative selection. Alleles that helped them survive or sped up their growth experienced positive selection. Each drop that Lenski drew out of each flask reflected these changes in the frequency of alleles.

Every 500 generations, Lenski stored some of the bacteria from each of the 12 lines in a freezer. The samples became a frozen fossil record that he could resurrect later. Once Lenski thawed out the bacteria, they could grow just as well as they had before. Lenski and his colleagues would put the ancestral bacteria in a petri dish with some of their descendants and watch them grow together. By observing these lines of bacteria under identical conditions, Lenski could directly measure how much their fitness changed over the course of the experiment. **FIGURE 6.11** shows the evolution of Lenski's *E. coli* over the first 20,000 generations. (The experiment has now progressed for 50,000 generations.) The average fitness of the 12 populations increased by approximately 75 percent relative to the ancestor (Ostrowski, Robert, and Lenski 2008).



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The bacteria in Lenski's experiment have experienced natural selection. New mutations have caused the descendants to reproduce faster under the conditions of the experiment than their ancestors did. (Adapted from Cooper and Lenski 2000.)

The increasing fitness of the bacteria was the result of new mutations that arose in each line. When Lenski started the experiment in 1989, pinpointing such new mutations was difficult, costly, and time-consuming. Now it's possible to sequence the entire genome of a microbe for a few hundred dollars. That falling cost has allowed Lenski and his colleagues to pinpoint the mutations that natural selection has favored—as opposed to mutations that have lowered their fitness or had no effect at all.

They've found a number of beneficial mutations. In one line of bacteria, for example, they found a single base that had changed since the original *E. coli*. The mutation was located in a gene control region known as *BoxG1*, which regulates two nearby genes. The genes, called *GlmS* and *GlmU*, encode proteins that help synthesize the bacterial cell wall (<u>Stanek, Cooper, and Lenski 2009</u>). To confirm that the mutation actually increased the bacteria's fitness, they engineered the ancestral bacteria so that it had the new version of *BoxG1*. That single base raised the relative fitness of the bacteria by 5 percent.

Lenski and his colleagues then set out to trace the origin of the mutation. At some point during the evolution of the bacteria, they hypothesized, the mutation must have emerged spontaneously. They turned to the line's frozen fossil record and thawed bacteria from each 500-generation sample. They then searched for the earliest appearance of the *BoxG1* mutation.

None of the bacteria they examined from generation 500 had the *BoxG1* mutation, indicating that it must have arisen later. The bacteria in generation 1000 told a different story: 45 percent of them carried the mutation. And in generation 1500, the researchers found that 97 percent of the bacteria had it. This rapid spread is the kind of pattern you'd expect from a mutation that arose between generations 500 and 1000 and allowed bacteria to reproduce faster than competitors that lacked it.

Lenski and his colleagues are now trying to figure out how the BoxGI mutation raises $E.\ coli\,\dot{s}$ fitness. Bacteria with the BoxGI mutation produce fewer GlmS and GlmU proteins. It's possible that these bacteria are investing

fewer resources into building their membranes. They can then redirect this energy to other functions that speed up their reproduction.

It's intriguing that the *BoxG1* mutation evolved in only one out of the 12 lines of *E. coli* in Lenski's lab. Other mutations, by contrast, have arisen independently in several different lines. Lenski and his colleagues have even identified three genes that mutated in all 12 lines. While evolution moved in the same overall direction in their experiment—a rapid increase in fitness followed by a tapering off—Lenski has found that the bacteria got there with different combinations of mutations.

Selection in the Balance

Lenski's bacteria reproduce asexually, dividing in two and bequeathing their genome to both daughter cells. Sexually reproducing organisms, on the other hand, merge their genes. As a result, there are some important differences between the way natural selection acts on asexual organisms and sexual ones.

When a male and a female organism mate, for example, they can combine their beneficial mutations in a single offspring. That offspring is at a greater competitive advantage than an offspring with the mutations from just one parent. Sexual reproduction also means that each offspring gets two copies of each gene. Different combinations of alleles can alter the fitness of an organism—and sometimes in unexpected ways.

Recall the oddly low frequency of SS carriers in Nigeria (Figure 6.3). It turns out that the S allele gives rise to a deformed hemoglobin molecule. The red blood cells that carry them are deformed as well, taking on a long, curved shape like the blade of a sickle. This deformity leads to a dangerous condition known as sickle-cell anemia. Many of their red blood cells die. Others clump together, damaging blood vessels, organs, and joints. Without medical treatment, sickle-cell anemia is often fatal. Sickle-cell anemia is the reason that SS carriers are out of Hardy-Weinberg equilibrium: some people with sickle-cell anemia die before they can have children. As a result, the S allele experiences strong negative selection.

At the same time, Cavalli-Sforza and his colleagues also found fewer people in Nigeria with the AA genotype than if the population was in Hardy—Weinberg equilibrium. They also found more people than expected with the AS genotype. That's because the S allele has more than one effect on our phenotype. Besides causing red blood cells to sickle, it also protects people from malaria, a disease that kills 881,000 people a year and infects an estimated 247 million. It's also a disease that is especially prevalent in Nigeria.

Malaria is caused by a single-celled protozoan called *Plasmodium*, which is carried by mosquitoes. When a mosquito bites a victim, *Plasmodium* slips into the bloodstream and invades red blood cells. It transforms fibers inside

the cells into conveyor belts that deliver parasite proteins to the cell's surface. There, the proteins cause red blood cells to stick together. The clumps of cells clog blood vessels, sometimes leading to fatal bleeding. The S allele gets in the way of the parasite, due to the way it alters the hemoglobin molecule. Hemoglobin produced from the S allele cuts off the conveyor-belt fibers when it encounters them. As a result, *Plasmodium* can't deliver proteins to the cell's surface, and it thus causes fewer red blood cells to clump (<u>Cyrklaff et al. 2011</u>).

If the *S* allele had no malaria-protecting qualities, it would rapidly become very rare, because people with two copies of it would have fewer children. People with one copy of the *S* allele enjoy this protection from malaria while avoiding the potentially lethal sickle-cell anemia that can come with having two copies (FIGURE 6.12). They are more likely to survive long enough to have children, to whom they pass down both alleles. Natural selection thus favors a balance between the *A* and *S* alleles, rather than driving one of them toward fixation. Biologists call this special form of selection balancing selection.

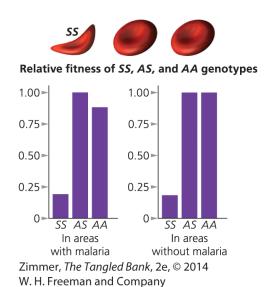


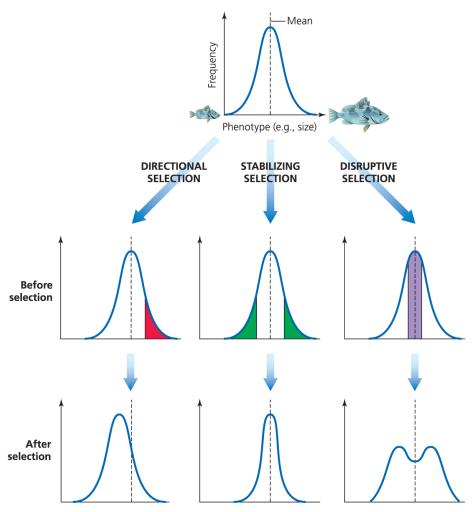
FIGURE 6.12

People who carry one copy of the *S* allele are more likely to survive malaria than *AA* homozygotes. People who are *SS* homozygotes, on the other hand, suffer sickle-cell anemia and have much lower fitness. The higher fitness of *AS* heterozygotes has the unfortunate effect of raising the frequency of *SS* homozygotes in the population.

Sickle-cell anemia drives home a sobering truth about the nature of fitness: fitness is not an inherent quality. It emerges from the relationship of organisms to their environment. If malaria were eliminated tomorrow, the AS genotype would immediately lose its relative fitness, and the S allele would begin to disappear.

The Speed and Direction of Evolution

The case of sickle-cell anemia gives us a glimpse at the variety of forms that natural selection can take. FIGURE 6.13 shows some of the other patterns biologists have documented. Each graph shows a phenotypic trait—the body size of fishes in a pond, for example—and how that trait influences reproductive success. In directional selection, the fishes at one end of the curve reproduce more than the others. In stabilizing selection, the fishes with body sizes within a certain range have more offspring than fish outside of it. In disruptive selection, the fishes with the average body size experience negative selection, while fishes further from average reproduce more.



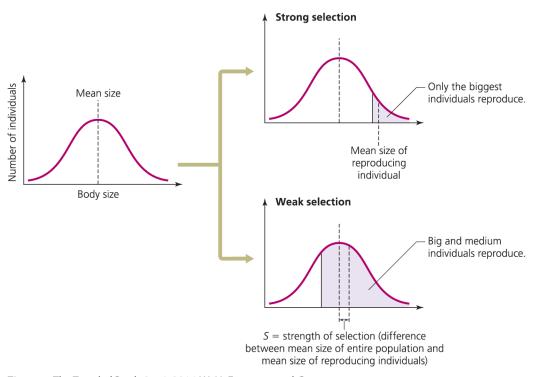
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FIGURE 6.13

Directional selection (left) favors individuals at one end of the distribution of a trait. This graph shows selection on the size of fishes. Here, large individuals have lower fitness than smaller individuals (negative selection against large sizes is indicated by the red shading). Stabilizing selection (middle) favors individuals with a trait near the mean of the population. In this case, fish with the largest and smallest body sizes have the lowest fitness (green shading). Stabilizing selection can narrow the range of body sizes. Disruptive selection (right) selects against the population mean, favoring individuals at either end of the distribution (purple shading). If disruptive selection is strong enough, it can turn the single peak of this graph into two peaks, one for smaller fishes and another for bigger ones.

Whatever form natural selection takes, a population will respond to it by evolving rapidly, slowly, or not at all. The speed of its response depends on four key factors: the strength of selection, the amount of variation in the population, how much of that variation is inherited, and the size of the population.

The strength of selection reflects the difference between the mean value of a trait in an entire population and the mean value of that trait among the individuals that reproduce (FIGURE 6.14). Let's say that a drought hits a pond and only big fish can survive. Selection is strong if only the very biggest fish survive. In a milder drought, smaller fish might be able to survive and reproduce as well. In that case, selection is weaker.



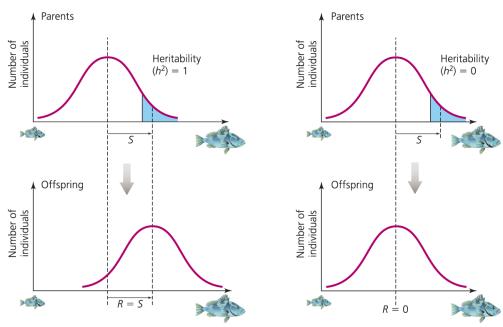
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FIGURE 6.14

The graph on the left shows the range of body sizes in a hypothetical population of fishes. Top right: If the population experiences strong selection for large body size, only the very biggest individuals reproduce. The mean size of the reproducing individuals is much bigger than the mean of the entire population. Bottom right: If selection is weak, big and medium individuals reproduce, and the mean size of the reproducing individuals is much closer to the mean size of the entire population.

What will the offspring of these selected fish look like? The answer depends on how much of the variation in their body size is due to their genetic variation and how much is due to environmental factors. Let's consider the two most extreme possibilities. If body size is purely the result of environmental factors—the temperature of the water, for example, or how much food the fish larvae find—then the heritability of body size is zero. Even though only big fish are selected to reproduce, their offspring will have the same range of body sizes that the previous generation had. On the other hand, if the heritability equals one—if all the variation is entirely due to genetic factors—then the big fish will pass down their size-related alleles, and the average size of fish in the population will increase.

The interplay between these factors can be summed up in an elegantly brief equation: the response of a population is the product of the heritability of a trait times the strength of selection. If selection is strong, a population can respond with a rapid change, even if a trait is only weakly heritable. And even weak selection can lead to significant evolutionary change, as long as a trait's heritability is high (FIGURE 6.15).



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Left: In this population, body size is a completely heritable trait. If the population experiences strong selection (S) for body size, the next generation's mean body size increases dramatically. The response (R) to selection is equal to the selection. Right: In this population, body size has no heritability. In other words, the size of parents is not correlated with the size of their offspring. Even if the population experiences strong selection for body size, the mean size does not change in the next generation. The response is zero.

Evolution in Our Own Time

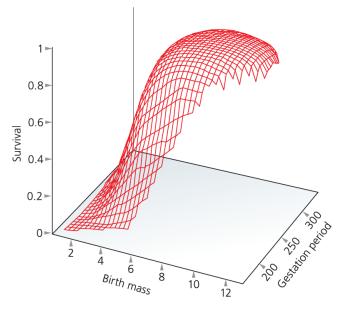
Charles Darwin managed to visit only a handful of the Galápagos Islands in 1835 while on his journey aboard the HMS *Beagle*. Among the many islands the ship passed by was a tiny volcanic cone known as Daphne Major. Even today, it is not an easy place to visit. To set foot on Daphne Major, you have to approach a steep cliff in a small boat and then take an acrobatic leap onto a tiny ledge. There are no houses on Daphne Major and no supply of water. In fact, just about the only things to see on Daphne Major are low, scrubby plants and the little birds that eat their seeds.

Box 6.1

Mapping the Fitness Landscape

In most of the studies we look at in this chapter, scientists measure the fitness of just one trait in a population. We can represent those measurements on a simple x-y graph, where the x-axis represents the value of the trait, and the y-axis represents the fitness of that value. But the reproductive success of an organism depends on many traits, not just one. To represent the effects of two traits on fitness, we have to switch to a three-dimensional graph.

FIGURE 1 shows one such graph, produced by Dolph Schluter and Douglas Nychka, that shows the fitness of human babies. Schluter and Nychka started by examining medical records from 7307 babies. For each child, they compared two traits—how long their mother carried them before giving birth, and how much they weighed when they were born. As a measure of fitness, the researchers looked at the records to see whether the babies survived their first two weeks.

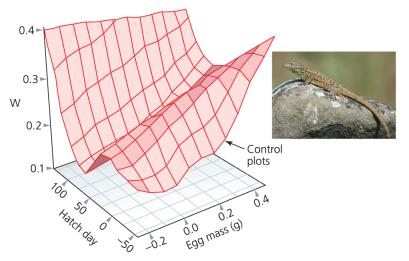


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FIGURE 1 A fitness landscape for human babies. (Data from Schluter and Nychka 1994.)

From these data, Schluter and Nychka created a "fitness landscape" that looks like a dome-shaped hill. It shows that selection acts most strongly against small babies with short gestation periods. Babies who have intermediate values for both traits have the highest fitness. The dome-shaped topography tells us that infants with different combinations of gestation and birth weight may still have the same odds of surviving. A baby with low birth weight and a gestation of moderate length, for example, has the same fitness as a heavier baby that was born sooner (Schluter and Nychka 1994).

FIGURE 2 shows a very different landscape. This one represents the fitness of side-blotched lizards in California. Erik Svensson and Barry Sinervo wondered how the mass of eggs and the timing of their hatching affected the survival of the lizards (Svensson and Sinervo 2000). They manipulated lizard eggs to make them larger or smaller than average. The scientists found an overall trend of higher fitness for bigger eggs. They also found that early- and late-hatching lizards were more likely to survive than lizards born in the middle of the hatching season. Svensson and Sinervo found that the cause of this trough is competition from other newly hatched lizards.



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: Selective loss of polymorphic mating types is associated with rapid phenotypic evolution during morphicspeciation by Corl, David, Kuchta and Sinervo. PNAS, February 16, 2010.

FIGURE 2 A fitness landscape for side-blotched lizards. The values for hatch day are relative to the average hatching day; the values for egg mass are relative to the average egg mass. Wis a measure of fitness. (Data from <u>Svensson and Sinervo 2000</u>.)

These two studies demonstrate that two traits can produce a much more complex fitness landscape than one. A complete landscape would have to include many other traits—producing a multidimensional space that's beyond our ability to visualize.

In 1973, a British-born couple named Peter and Rosemary Grant came to Daphne Major and spent months on the island (FIGURE 6.16). They've returned every year since, bringing a team of students with them, along with all the supplies they need for a lengthy stay: tents, coolers, jugs of water, cooking fuel, clothes, radios, binoculars, and notebooks. This dedication has allowed the Grants—who are now biologists at Princeton University—to make one of the most extensive studies of natural selection in the wild.



Left to right: Peter and Rosemary Grant; Peter and Rosemary Grant.

FIGURE 6.16

Peter and Rosemary Grant catch Darwin's finches and take measurements of their bodies. The tiny island of Daphne Major, which is accessible only by scrambling up the surrounding cliffs, provides an unusually pristine environment for this study.

The Grants can't consult a perfect genealogy of all the birds that ever lived on the island. They cannot thaw out birds that lived thousands of generations ago to compare them to their living descendants. Nevertheless, with enough tenacity and patience, they can document the process that Darwin and Wallace first proposed over 150 years ago.

There are 13 species of Darwin's finches on the Galápagos Islands, but one species has captured most of the Grants' attention: the medium ground finch (*Geospiza fortis*), a seed-eating bird. The Grants chose to study the population on Daphne Major despite the inaccessibility of the island—indeed, precisely because of it. It remains relatively pristine. No one has ever tried to farm on the island. No one introduced goats or other invasive species. As far as the Grants can tell, no species on Daphne Major have become extinct since the arrival of humans.

The island has the added advantage of being ecologically simple. There aren't very many plant species on Daphne Major, so the Grants were able to identify and measure every type of seed that the island's finches eat. The island is small, and so is its population of birds. On Daphne Major only a few hundred ground finches may be born in a given year, and most spend their entire lives there, thus permitting the Grants to mark and follow every

individual in the population. Emigrant finches rarely leave the island, and immigrants rarely arrive. As a result, the Grants can be confident that migrations have a negligible effect at best on the changes in the allele frequencies of the island population.

Each year, the Grants survey every medium ground finch on Daphne Major, measuring vital statistics such as their body mass and beak width (FIGURE 6.17). They trace families, determining how many offspring each bird had, and how many offspring their offspring had. From year to year, the Grants also compare individual finches to their offspring to determine how strongly inherited each kind of variation is.

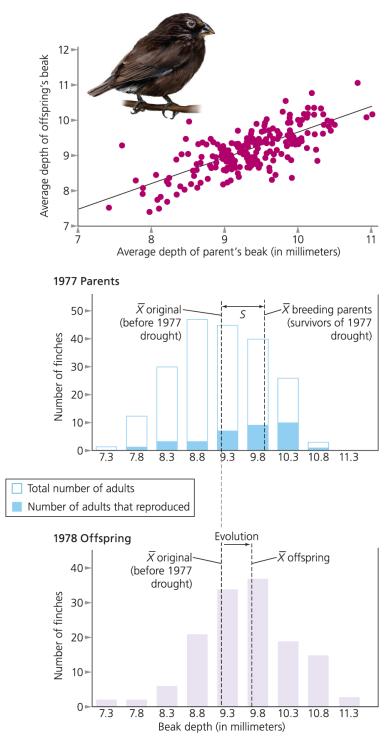


Peter and Rosemary Grant.

FIGURE 6.17

Medium ground finches on Daphne Major differ in the thickness of their bills. This variation causes some individuals to be more efficient at processing hard seeds.

The Grants and their team found big-beaked birds tend to produce chicks with big beaks, and small-beaked birds tend to produce chicks with small beaks (FIGURE 6.18). In other words, the variation of the beak is strongly influenced by genes. It could thus potentially evolve rapidly in response to natural selection.



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FIGURE 6.18

Top: The size of beaks in medium ground finches is heritable. In other words, birds with big beaks tend to have offspring with big beaks, too. Middle: During a drought in 1977,

birds with large beaks had more offspring on average than other birds. (The white bars show the total number of medium ground finches on Daphne Major with beaks in each size class, before the drought. The blue bars show the number of birds with beaks in each size class that survived the drought and subsequently reproduced.) Bottom: The average beak size increased in the offspring produced by birds surviving the drought. The dashed vertical lines show the average bill size from one year to the next. (Data from Grant and Grant 2002.)

The Grants reasoned that the size of a bird's beak could affect how it ate seeds, so they investigated the kinds of food available to the birds on Daphne Major. They measured the sizes and hardness of each of the seeds produced by two dozen species of plants on the island. They dug up soil samples and counted all of the seeds contained in them. The Grants and their colleagues also closely observed the birds as they ate, noting which kinds of seeds they chose and the time it took birds to process seeds of each type. During that first season alone, they observed over four thousand meals.

When the Grants started their historic study, they were surprised to find that different species did not specialize on different kinds of seeds. In addition to the medium ground finch, Daphne Major is also home to the small ground finch (*Geospiza fuliginosa*), which has a narrower, pointier bill. Despite the different shapes of their beaks, both species of birds fed on the same soft, small seeds that were abundant on the island. Even species that weren't seed specialists, such as cactus finches, were eating the seeds.

When the Grants returned six months later, however, the island was transformed. The dry season had begun, and the island had not gotten a drop of rain for four months. Many of the plants on Daphne Major had died, leaving behind a barren landscape. The small, soft seeds were all gone. Now the birds were no longer all eating the same kind of food. They had become specialists. The Grants discovered that even within each species, individuals selected different kinds of seeds. Their choice, it turned out, depended on subtle differences in the shapes of their beaks.

The medium ground finches could choose from two kinds of seeds: small seeds from a plant known as spurge (*Chamaesyce amplexicaulis*) and hard, woody seeds from the plant *Tribulus cistoides*, commonly called caltrop. Finches with big beaks (11 millimeters deep) could crack open the caltrop

seeds in 10 seconds. Finches with beaks 10.5 mm deep needed 15 seconds. If a bird's beak was 8 mm deep or less, it took so long to crack caltrop seeds that the bird gave up on it altogether. Instead, it ate only small spurge seeds.

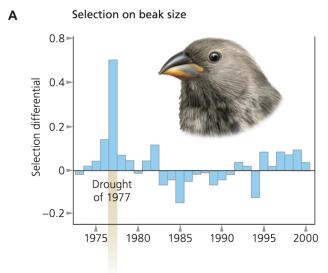
The Grants found that the size of a finch's beak could make the difference between life and death. In 1977, Daphne Major was hit by a major drought. Most of the spurge plants died, leaving the medium ground finches without any small seeds to eat. Many of the birds died, most likely because they couldn't crack open the big seeds from caltrop. The Grants discovered that within a few years, the population of finches had recovered. But now the average size of their beaks was deeper. Before the drought, the population ranged in beak size from 8 to 11 mm with an average depth of 9.2 mm. After the drought, the average beak size had shifted half a millimeter to 9.7 mm, or about 15 percent of the range of variation (Grant 1986).

The shift occurred because finches with bigger beaks had a better chance of surviving the drought. They could therefore produce a bigger fraction of the next generation. In other words, natural selection caused the average size of the beaks of medium ground finches to increase within the population.

Five years later, the Grants were able to observe natural selection at work again. At the end of 1982, heavy rains came to the islands. Spurge bloomed, producing an abundance of small seeds. Now small-beaked birds had the advantage. They could eat small seeds more efficiently than the big-beaked ones, allowing them to grow faster and have more energy for producing offspring. In just a few generations, the average size of beaks decreased by 2.5 percent (about a tenth of a millimeter).

The Grants had made a historic observation. It was the first time scientists had measured the effects of natural selection in a wild population as they were unfolding. The Grants had measured the heritability of beak size, they had measured the strength and direction of several episodes of natural selection acting on beak size, and they had measured what happened to the population for several generations after the episode of selection.

But the Grants did not abandon Daphne Major after their initial observations. They've continued coming back for nearly 40 years (FIGURE 6.19). That persistence has paid off impressively. Their research now offers some deep insights into how natural selection works (Grant and Grant 2002).



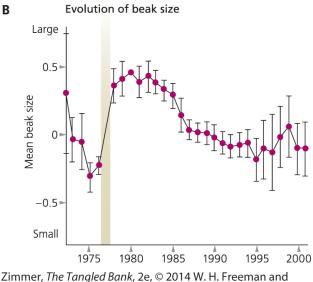


FIGURE 6.19

Company

Over the past four decades, the strength and direction of natural selection on beaks have fluctuated in response to the changing food supply. A: The selection differential shown in this graph is the difference between the mean beak size of the population and the mean beak size of the individuals producing offspring in the next generation. In some years, birds with large beaks were favored (bars above the horizontal line); in other years, birds with small beaks were (bars below horizontal line). In still other years, selection on beak size was minimal. B: The finch population evolved in response to these episodes of selection, with the result that beak size fluctuated in tandem with the directions of selection. (Data from Grant and Grant 2002.)

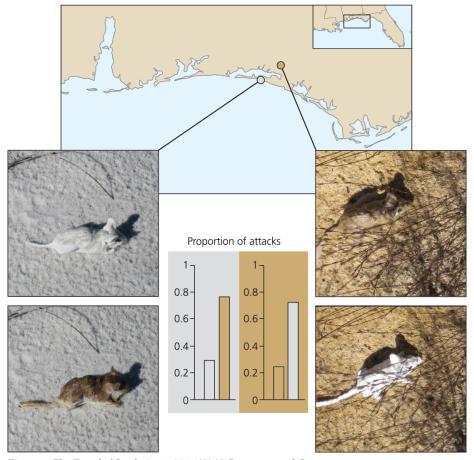
One important lesson from those four decades of research is that natural selection may itself be variable. Had the Grants sampled Daphne Major only during the wet seasons, they would have missed important episodes of directional selection. It was during the dry seasons—and in particular, the dry seasons of drought years—that selection favored big beaks. Both the strength of selection, and its direction, fluctuated several times over the course of their study.

A second lesson is that evolution can occur with surprising speed. Before the Grants conducted their research, many evolutionary biologists maintained that evolution likely occurred over very long timescales. Gradual changes in the fossil record unfolded over millions of years, suggesting that the strength of natural selection was probably very weak—weaker, for example, than the artificial selection imposed on populations of domesticated plants and animals. But the Grants were able to observe evolutionary changes in a natural population that were every bit as fast as those resulting from artificial selection. The selection they measured was strong, and their populations evolved in a matter of generations.

Mutations on the Beach

The studies of Peter and Rosemary Grant inspired hundreds of other evolutionary biologists to head out into the wild and measure natural selection. They've documented the marvelous complexity of natural selection, and, in cases such as Hopi Hoekstra's work on oldfield mice, they have zeroed in on the specific genes that selection is altering.

When Hoekstra started studying oldfield mice, she hypothesized that their light and dark coats evolved as protection against predators. The predators would have a hard time seeing mice that blended well against their background. To test this hypothesis, Hoekstra and her colleague, Sacha Vignieri, then at Harvard, conducted an experiment. They made hundreds of life-sized clay models of mice and scattered them on a Florida beach. Half of the imitation mice were dark and half were light. Hoekstra and Vignieri then waited for predators to attack the models (FIGURE 6.20).



Zimmer, The Tangled Bank, 2e, $\ \odot$ 2014 W. H. Freeman and Company Photo: Sacha Vignieri.

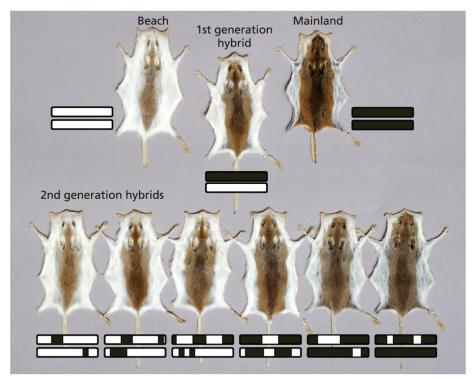
FIGURE 6.20

Hopi Hoekstra and her colleagues set up an experiment to test how the color of oldfield mice influenced their chances of being attacked by predators. They painted clay mouse models to resemble beach or mainland forms and placed the models in either mainland or beach habitat in Florida. Blending into the background reduced predation rate in both the beach and mainland habitat. Predation rates of dark clay models in beach habitats (left) were much higher relative to white models, and predation rates of light models in mainland habitats (right) were much higher relative to dark models. Photos: Sacha Vignieri. (Information from Vignieri et al. 2010.)

Birds and foxes attacked some of the imitation mice, but then quickly discarded them. Hoekstra and Vignieri gathered all the models and tallied the ones that had been damaged by predators. The dark models, they found, were much more likely to be attacked than the light ones. When the scientists

repeated the experiment on dark soil, they got the opposite result (<u>Vignieri</u>, <u>Larson</u>, and <u>Hoekstra 2010</u>).

Hoekstra and Vignieri's experiment showed that different colors could be selected in each habitat. But how did the variation in the color of the mice arise in the first place? To find out, Hoekstra and her colleagues used quantitative trait locus analysis (see <u>page 120</u> for details of this method). Hoekstra and her colleagues interbred three pairs of light and dark mice, and then they reared two additional generations. The 465 mice in the third generation developed a range of coat colors, grading from nearly white to mostly brown (<u>FIGURE 6.21</u>).



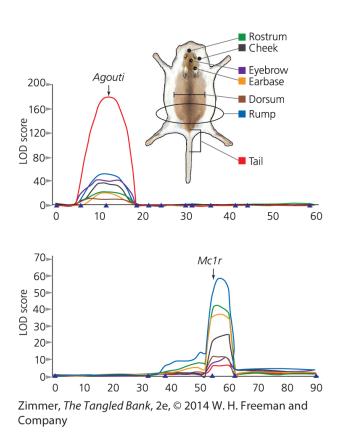
Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company From "Adaptive Variation in Beach Mice Produced by Two Interacting Pigmentation Genes" by Steiner, Weber and Hoekstra. PLos Biology 08/14/2007.

FIGURE 6.21

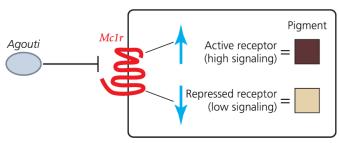
To study the evolution of color in oldfield mice, Hopi Hoekstra and her colleagues carried out a breeding program. They began by crossing white beach mice with brown mainland mice. The hybrid offspring inherit one copy of each chromosome from their parents, and they have intermediate coat color. The scientists then bred these hybrids to produce a second generation. Each of the bred mice inherited different combinations of genetic

regions from the original mice. Hoekstra and her colleagues were then able to search the mice's genomes for genetic markers that tended to be associated with either light or dark coat color.

Hoekstra and her colleagues scanned the DNA of the mice, looking for segments that showed up most often in light mice and least often in dark ones. Two genes, known as *Agouti* and *Mc1r*, turned up frequently (FIGURE 6.22). This discovery makes sense, because *Mc1r* encodes a receptor that sits on the surface of a cell, where it can pick up signals instructing the cell to produce the pigment melanin. *Agouti* encodes a protein that interferes with that communication. High levels of Agouti proteins lead to light hairs; less Agouti leads to dark ones (FIGURE 6.23). Beach mice turn out to have mutations in both *Agouti* and *Mc1r*. The *Agouti* mutation leads to the production of more of the protein. The *Mc1r* mutation, on the other hand, makes it less sensitive to signals (Steiner et al. 2009).



When Hopi Hoekstra and her colleagues inspected the genomes of their hybrid mice, they found two locations that were strongly correlated with the coat color. Each location contains a color-influencing gene, known as *Agouti*, and *Mc1r*. (Figure data from <u>Steiner et al. 2007</u>.)



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FIGURE 6.22

Mc1r encodes a receptor that triggers cells to produce pigment. Oldfield mice have two alleles for the gene, one that responds strongly to signals and one that responds weakly. *Agouti* also influences coat color by repressing *Mc1r*, so that it can't trigger the production of pigment. These two genes have evolved through natural selection so that oldfield mice blend into their habitats.

Experiments like these allow Hoekstra to build a detailed hypothesis for the evolution of coat color in oldfield mice—a hypothesis that can encompass the genetic basis of the evolution and the ecological factors that drive natural selection. In mainland populations, alleles for genes like *Agouti* and *Mc1r* create variation in the color of the mice. Any mice that end up with alleles that produce even a moderately light coat tend to get picked off by predators, because they're so easy to see against the dark background of the soil. The darker mice survive and pass down their alleles.

Several thousand years ago, barrier islands emerged on the coast of Florida, creating a new kind of habitat. The oldfield mice expanded into it, and there a dark coat became a liability, not a benefit. Lighter mice were less likely to be killed because they blended better into the background of white sand. The alleles that fostered light fur became more common—including a mutation to *Mc1r* that made cells less sensitive to pigment signals, and

another to *Agouti* that expanded the area where melanocytes developed slowly.

Hoekstra's research illustrates an important benefit of studying natural selection. As she investigates how mice evolved white coats, she has ended up discovering previously unknown things about the biology of color, such as the way *Agouti* pre-patterns embryos. And these insights can shed light on other species. Melanocytes are responsible for pigments in bird feathers (page 50), squid ink, and even human skin. Our skin pigment protects us from ultraviolet radiation from the sun, which can damage DNA, and as a result, some variants of *Mc1r* can increase the risk of skin cancer. Scientists are now performing experiments on mice to understand this link (Beaumont, Liu, and Sturm 2009). Evolution has also provided researchers with a natural experiment on the coast of Florida.

The Rise of Milk

If your ancestors hail from Western Europe, chances are you can digest milk. If you're Chinese, chances are you can't. It turns out that the difference is partly the result of natural selection on humans over the past few thousand years (FIGURE 6.24).





Left: Ton Koene/age fotostock/Superstock; Right: Bernard/imagebroker/Superstock.

FIGURE 6.24

Humans domesticated cattle in both East Africa (left) and Northern Europe (right) several thousand years ago. In both places, mutations arose that enabled humans to digest milk as adults. These mutations spread rapidly because they raised fitness.

Humans are mammals, and a hallmark of living mammals is that mothers produce milk for their young. To digest the lactose sugar in milk, young mammals produce an enzyme in their intestines called lactase. Around the time young mammals are weaned, they typically stop producing lactase. Since they're no longer drinking milk, producing the enzyme becomes a waste of energy that could be directed to more rewarding ends.

Humans are odd in this respect: over 2 billion people continue to produce lactase as adults. As a result, they can continue to harvest the energy in milk.

The other 70 percent of humans are like other mammals: they stop producing lactase after they are weaned. If they try to drink milk or eat ice cream as adults, they can't break down the lactose. Bacteria in their intestines that produce lactase enzymes feed on the lactose instead. They give off gas and other wastes that cause indigestion.

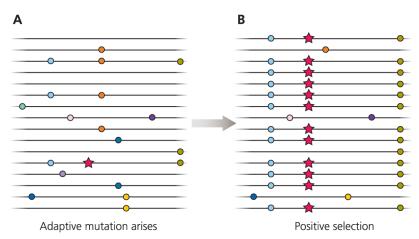
The ability of so many people to tolerate lactose as adults is the result of human natural selection over the past few millennia. Starting about 10,000 years ago, humans began to domesticate cattle in a few regions of the world, such as northwest Europe and East Africa. These humans' diet changed drastically as a result: they could now eat energy-rich milk, yogurt, and other milk-based foods. In these regions, mutations enabling lactose to persist through adulthood were favored, since their bearers could take full advantage of these new food sources.

We can see the fingerprints of this natural selection in living humans. The gene for lactase (known as LCT) has mutated into the lactose-tolerant form in societies that are traditional cattle herders. (The new allele is called *LCT*P*.) In Europe, *LCT*P* is most common in northwest Europe, where cattle herding originated, and rarest in southeast Europe, the furthest point from that origin. Within countries, the societies that consume milk as a traditional food have a higher frequency of *LCT*P* than the societies that don't rely on milk (Swallow 2003).

Geneticists have also found that the *LCT*P* allele has evolved at least twice: lactose-tolerant Europeans and East Africans have different mutations in the *LCT* gene. The finding that two different mutations to the same gene arose in separate populations and then became so common also argues strongly for natural selection.

The recombination of chromosomes provides yet another line of evidence that natural selection has favored LCT*P. To understand how it does this, imagine that you're cutting a new deck of cards. After the first cut, most cards will still be nestled among their original neighbors. With each additional cut, those original sequences get smaller and smaller. Eventually, the deck ends up randomly mixed. Sexual recombination shuffles DNA in a similar way, by swapping segments between pairs of chromosomes. At first, neighboring alleles will stay together. But over time, the alleles will become randomly mixed between the two chromosomes.

Strong selection can sweep chunks of DNA through to fixation faster than recombination breaks them apart. An allele that produces a big increase in reproductive success will spread rapidly through a population, and it will carry along its neighboring DNA. As a result, many individuals will share large segments of DNA that surround an allele experiencing natural selection. In both European and East African populations, *LCT*P* has this hallmark of strong selection (<u>Tishkoff et al. 2007</u>; **FIGURE 6.25**).



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FIGURE 6.25

During recombination, matching segments of DNA get swapped between chromosomes. Two alleles sitting next to each other on the same chromosome can thus become separated on different ones. Over many generations, this shuffling creates many different combinations of alleles. Natural selection, however, can slow down this shuffling. A: Each line represents a segment of DNA of one individual in the population. Circles represent nucleotide bases unique to that individual. A new mutation (red star) arises in one individual and raises its fitness. B: The same population, a number of generations later. Individuals who inherited a segment of DNA with the new mutation had higher fitness. The mutation increased in frequency, carrying along its neighboring DNA. As a result, this particular recombinant will be unusually abundant in the population. Scientists have found this unusual pattern surrounding mutations in a gene called *LCT*, which allows people to digest milk as adults.

Human-Driven Evolution

In the 1960s, the government of France had a plan. To attract tourists to the Mediterranean coast, they would build entire cities from scratch. There was just one problem: the balmy climate along the coast created a splendid environment for mosquitoes (*Culex pipiens*) to breed (<u>FIGURE 6.26</u>). Before the tourists came, the mosquitoes would have to go.



FEMA/Getty Images.

FIGURE 6.26

A single allele can allow mosquitoes to resist insecticides. Such resistance alleles have spread rapidly in mosquito populations around the world, due to natural selection.

The government launched a program to regularly spray mosquito-breeding sites along the coast with pesticides. Starting in 1969, they used organophosphate insecticides, which kill the insects by blocking an enzyme called acetylcholinesterase (AChE1) in the mosquito nervous system.

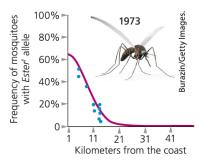
At first, the treatment was successful. The mosquito population fell, and people got bitten less often. But in 1972, the mosquitoes started coming back.

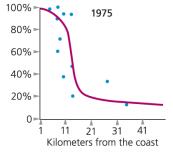
To find out why they were rebounding, Nicole Pasteur of the University of Montpellier collected mosquito larvae from streams and cisterns. She took the insects to her lab to expose them to different doses of insecticides.

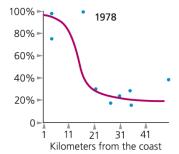
The mosquitoes from just north of the spray zone died when she exposed them to even a weak dose of insecticide. But when Pasteur ran the same experiment on mosquitoes from the coast, where spraying was heavy, the insects could resist stronger doses (<u>Pasteur and Sinégre 1975</u>).

Further research revealed the source of this resistance. *C. pipiens* produces an enzyme known as esterase that breaks down a wide range of toxins, including organophosphate insecticides. The esterase normally produced by a mosquito is not enough to eliminate the insecticide, and so the insect dies. Pasteur and her colleagues discovered that the resistant mosquitoes carried a mutation that increased the amount of esterase they produced. Mosquitoes carrying this allele, which was known as *Ester*¹, could eliminate the insecticide before it could kill them. They survived and reproduced, passing down the *Ester*¹ allele to their offspring (<u>Raymond et al. 1998</u>).

The scientists began conducting annual surveys around southern France to find more mosquitoes carrying $Ester^{l}$. Before 1972, they detected no $Ester^{l}$ alleles whatsoever. But by 1973, the allele shot up to a frequency of 60 percent in coastal populations. Farther inland, the allele was rarer; just 20 kilometers from the ocean, only 20 percent of the mosquitoes had it. As the years passed, the frequency of the $Ester^{l}$ allele in the coastal populations continued to climb until it reached 100 percent in 1978 (**FIGURE 6.27**).







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These graphs show the spread of the *Ester*¹ allele through mosquito populations around Marseilles. The *x*-axis represents distance from the Mediterranean Sea. By 1973, the *Ester*¹ allele was present in over half of the mosquitoes along the coast but was absent 21 kilometers inland. By 1976, it was fixed in the coastal populations but still rare inland. In 1978, the allele had become somewhat more common inland but still nowhere as prevalent as on the coast. This difference in selection was due to heavy pesticide spraying on the coast, raising the fitness of *Ester*¹ alleles there. (Data from <u>Raymond et al. 1998.</u>)

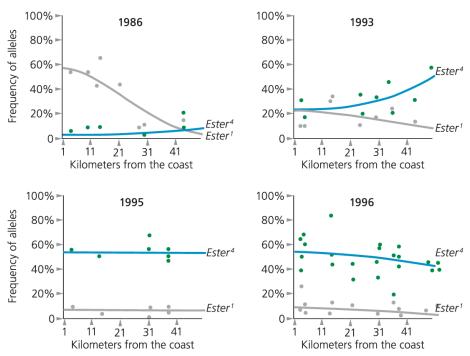
The evolution of insecticide resistance took different paths on the coast and farther inland due to an important feature of genes: they usually perform more than one function. A mutation that boosts one function may interfere with another. When the $Ester^{I}$ allele emerged in the early 1970s, it provided mosquitoes with resistance to insecticides, but it had other effects on the mosquitoes. Researchers at the University of Montpellier have found that the $Ester^{I}$ mosquitoes have a higher probability of being caught by spiders and other predators than do insecticide-susceptible mosquitoes (Berticat et al. 2004).

On balance, *Ester*¹ raised the fitness of coastal mosquitoes because they were exposed to so much insecticide. Even if the extra esterases make the mosquitoes more vulnerable to predators, they still, on balance, make the insects more fit.

Such is not the case farther inland, where there's no insecticide to resist. $Ester^{l}$ lowers the fitness of inland mosquitoes by making the insects easier prey. The curves in Figure 6.27 are the result of this shift in balance. This difference was maintained even as mosquitoes were migrating from one site to another and their genes were flowing across southern France. As soon as a mosquito carried an $Ester^{l}$ allele out of the insecticide zone, it was usually eliminated by selection.

As FIGURE 6.28 shows, the $Ester^I$ allele became common along the coast in the 1970s, but it later became rare. That's because a new allele, known as $Ester^4$, emerged around 1985. It also led to the overproduction of esterases. Intriguingly, $Ester^4$ became more common as $Ester^I$ was disappearing—even though it provides slightly less protection against insecticides. A clue to its

success comes from the shape of its curve. $Ester^4$ does not drop off steeply as you go inland. It's likely that $Ester^4$ does not impose the cost of $Ester^1$. Selection thus favors $Ester^4$ on the coast, but mosquitoes don't pay a price for carrying it if they migrate inland (Raymond et al. 1998).



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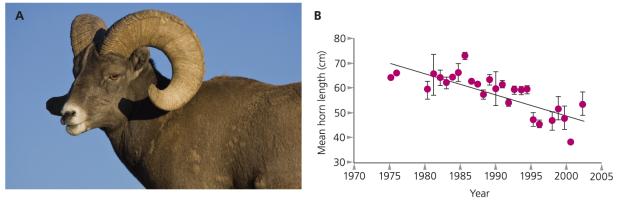
FIGURE 6.28

After spreading widely in the 1970s, the *Ester*¹ allele gradually became rarer in the 1980s and 1990s while another allele, known as *Ester*⁴, became widespread. The shift may reflect a physiological cost imposed on the insects by *Ester*¹. *Ester*⁴ alleles may confer resistance on mosquitoes without this cost, making its relative fitness higher and driving it to higher frequencies. (Data from <u>Raymond et al. 1998.</u>)

The selection that the French mosquitoes experienced was fundamentally similar to the selection that's been documented in Darwin's finches and old-field mice. But in one important respect, it's different. Natural conditions drove selection in the mice and birds. The mosquitoes, on the other hand, adapted to conditions created by human beings. They are just one of many species that are evolving in response to the increasing influence of humans.

Farmers, for example, have doused their crops in herbicides to kill weeds, which have also evolved resistance (<u>Powles and Yu 2010</u>).

Animals have evolved in response to hunting and fishing (<u>Allendorf and Hard 2009</u>). When trophy hunters go after bighorn sheep, they like to kill the males with the biggest horns. The same goes for deer, elk, and moose. By killing those individuals, hunters have driven the evolution of smaller antlers and horns (<u>FIGURE 6.29</u>; <u>Coltman et al. 2003</u>). Fishermen exert an even stronger selection on fish: In some salmon populations, 90 percent of the fish get caught. Fishermen tend to catch bigger individuals, and so the fish that reproduce tend to be small. As a result, many populations of fish have evolved in recent decades to a smaller size at maturity (<u>FIGURE 6.30</u>).



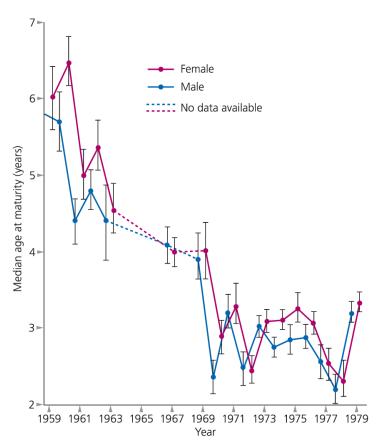
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FIGURE 6.29

A: Bighorn sheep (*Ovis canadensis*) have experienced selection from hunters who prefer large males with long horns. B: Over the past 30 years, this "unnatural" selection has resulted in the evolution of shorter male horns. (Data from <u>Coltman et al. 2003</u>.)







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Fisherman selectively harvest Atlantic cod (A), keeping only the largest fishes. In the process, they've created selection for smaller body size. After decades of this selection, the cod now reach sexual maturity at significantly smaller sizes (B). (Information from Beacham 1983.)

Human-driven natural selection is more than just a striking illustration of evolution in action. In some cases, it poses a direct threat to our well-being. The evolution of small fish threatens the sustainability of fisheries, for example, because small fish produce far fewer young than big ones do (Marteinsdottir and Begg 2002). Farmers worldwide spent over \$39 billion on pesticides in 2007, and the evolution of resistance in insects has led to the use of far stronger chemicals. These new pesticides can harm beneficial insects along with crop-eating species, and they can pose a risk to public health when they contaminate groundwater (Palumbi 2001). And, as we'll see in Chapter 15, human-driven natural selection has created a medical disaster: the rise of bacteria that are resistant to just about every kind of antibiotic.

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Molecular Evolution The History in Our Genes





Left to right: Top row: Shelly Perry/Getty Images; Mazdaguy03/Getty Images; LPETTET/Getty Images; ManoAfrica/Getty Images; Top middle: Klubovy/Getty Images; bobbidog/Getty Images; achirangshu/Getty Images; Dan Brandenburg/Getty Images; Bottom middle: SKashkin/Getty Images; azndc/Getty Images; Vikram Raghuvanshi/Getty Images; iofoto/Getty Images; Bottom: digitalskillet/Getty Images; sswartz/Getty Images; Vikram Raghuvanshi/Getty Images; azndc/Getty Images.

The 7 billion people on Earth today are physically and culturally diverse. But our DNA reveals a common heritage from early humans that lived in Africa some 150,000 years ago.

Sarah Tishkoff has been traveling from one end of Africa to the other for well over a decade. She took her first trip there as a graduate student in genetics at Yale University, and she still returns, now as a professor at the University of Pennsylvania. She has bounced along cratered roads in Tanzania, and she has traveled aboard hand-cranked ferries in the jungles of Cameroon. On her journeys, Tishkoff carries syringes, vials, and centrifuges. Her goal is to create a genetic portrait of the 1.033 billion people who live in Africa. She and her colleagues have gone a long way toward reaching that goal, having collected DNA from more than 7000 people from over 100 ethnic groups.



Sarah Tishkoff.

Sarah Tishkoff of the University of Pennsylvania crosses Africa to gather genetic samples to study human diversity.

There are many things Tishkoff hopes to learn from this portrait. She and her colleagues are beginning to identify alleles that make some Africans more vulnerable to certain diseases and resistant to others. But she also has come to Africa to understand history—not just the history of Africans, but the history of all humans. Tishkoff and her colleagues have created a detailed genealogy of the human race. The oldest fossil evidence of our species, *Homo sapiens*, is from East Africa and dates back to about 200,000 years ago (page 52). By studying the DNA of living people, Tishkoff has documented how humanity diversified in Africa for tens of thousands of years, after which a relatively small group of Africans migrated out of the continent, interbred with other hominin populations, and ultimately spread out across the rest of the world (Campbell and Tishkoff 2010).

In <u>Chapter 4</u>, we saw how scientists use anatomy to construct evolutionary trees. Until the 1990s, that sort of information was the only kind available to evolutionary biologists. But molecular biology has since provided them with a new way to study the tree of life. A

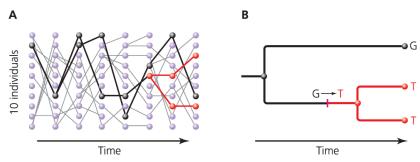
remarkable amount of history is encoded in the DNA of living species, and scientists are now using it to get answers to a range of questions about biology—from the origin of diseases to the functions of mysterious genes.

Genealogies of Genes

When you think of genealogy, you probably think of people—people like your father, your great-great-aunt, your fifth cousin once removed. You can trace your common ancestry with your relatives by tracing each person's ancestry. It turns out that your genes also have a genealogy. But it's different in some ways than your own.

To see why a gene's genealogy is different, imagine that you have a rare allele for a collagen gene. (Collagen is a stretchy protein in your skin and joints.) We compare the sequence of your allele to that of others and notice a mutation: at a position where most people have a guanine, your gene has a thymine. There are two ways to account for this pattern. One is that the mutation arose as the sex cells of your parents developed. The other is that one of your parents already had that rare collagen allele and passed it down to you.

Let's say that you got the collagen allele from your mother. Its presence in her genome can be explained in the same two ways. We might look back at your maternal grandfather's DNA and find the mutation there as well. If we had access to all your relatives' DNA, we could trace the allele back to its original mutation. And we could also move forward from that original mutation and trace the collagen allele's spread among your aunts, uncles, and cousins (FIGURE 7.2).



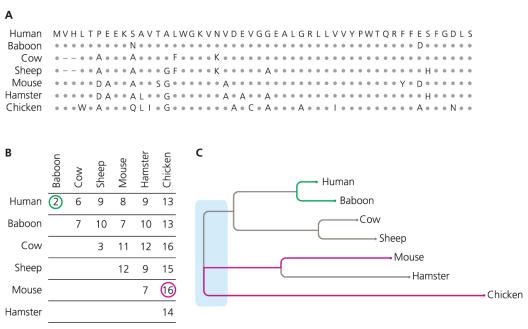
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A: This diagram shows a hypothetical population with ten individuals in each generation. Each individual is haploid, meaning it has only one copy of each gene. The dark lines show how one gene passes down from one generation to the next. In one line of descent, a base in the gene mutates from guanine to thymine (marked here in red). B: If we select three individuals in the youngest generation (high-lighted in A), we can create a phylogeny for the gene they share. The mutation from guanine to thymine serves as a marker, indicating that the two individuals that carry it are more closely related than either is to the third individual. (Data from Rosenberg and Nordborg 2002.)

Now imagine that we overlay this genealogy of the collagen gene on top of the genealogy of your flesh-and-blood family. They are not identical. Each time people have a child, they have only a 50 percent chance of passing down either copy of a gene. If you have a son and fail to pass down the rare collagen allele, it will disappear not just from him but also from all his descendants—unless, of course, those descendants acquire a copy from the other side of their family.

New alleles arise and spread through populations this way all the time. Over many generations, some alleles will disappear and others will spread to fixation, thanks merely to genetic drift. At the same time, populations become isolated from each other. As a result, new alleles that become common in one population can't spread to others. And over thousands of years, those populations diverge into separate species. (We'll take an indepth look at the evolution of new species in Chapter 10.) The result of all these processes is that different species will carry unique combinations of alleles. But they will also share some alleles with closely related species.

Scientists can uncover these relationships by comparing the DNA of different species and exposing evolution's signature on their genes. **FIGURE** 7.3 provides a simple example of this kind of analysis.



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Here's an example of how scientists use molecular data to reconstruct a phylogeny. A: The analysis starts with the amino acids in beta-globin protein. The human sequence is shown in full. The dots represent amino acids that are identical to the corresponding ones in the human version. The letters represent different amino acids. The dashes represent parts of the protein that are missing due to mutations that deleted that corresponding DNA. B: Scientists then tally the differences between each pair of species. C: Using a computer program, scientists find a tree that fits these differences best. Humans and baboons have almost identical versions of the protein. This finding reflects their close ancestry. The branches are different lengths to reflect the number of mutations that have accumulated in each lineage. This variation is illustrated in part C, which highlights two examples. Humans and baboons differ by only two positions, while mice and chickens differ by 16. The blue shading shows the parts of the phylogeny that scientists can't resolve with the data here. (Data from Hartl 2011.)

One of the big challenges of reconstructing phylogenies is not confusing convergently evolved traits for homologous ones. As we saw in <u>Chapter 4</u>, insects and birds may both have wings, but that's not a sign they evolved from a common winged ancestor. The same challenge holds true for using DNA to study phylogenies. If a collagen gene mutates from glycine to thymine

at the same site in two different lineages, the similarity of the two alleles doesn't mean they're closely related.

Molecular phylogenies are also challenging for another reason: gene trees are not always identical to species trees. Over time, new alleles will arise in a lineage; some will become fixed and some will be lost through drift. When species diverge, each new lineage will inherit some of those alleles, which will continue to evolve. The two processes—of speciation and the evolution of genes—do not happen in lockstep.

FIGURE 7.4 shows how gene trees and species trees can disagree. In Figure 7.4A, alleles of the gene present in species 1 and species 2 diverged more recently from one another than either did from the allele present in species 3. The same relationship holds true for the species themselves. In Figure 7.4B, on the other hand, the alleles and the species have different histories. The allele from species 2 happens to be more closely related to the allele from species 3.

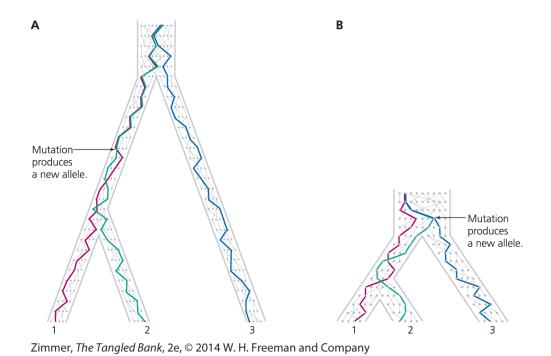


FIGURE 7.4

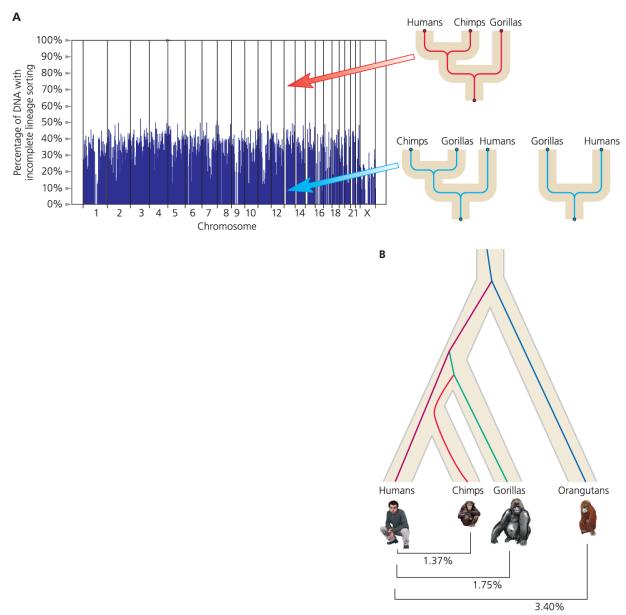
The genealogy of genes often reflects the phylogeny of species. A: If we look at two versions of the same gene in three species, we can trace their history back to ancestral genes. The common ancestor of the gene in species 1 and 2 is marked by "coalescence."

In this case, the relationship of the genes is the same as the species themselves. B: In a process called incomplete lineage sorting, the versions in 1 and 3 are more closely related. (Information from <u>Rosenberg and Nordborg 2002</u>.)

If we were to rely on this one gene alone to determine the phylogeny of the three species, we'd end up concluding that 2 and 3 are closely related sister species when, in fact, they are not. Scientists call this type of evolution incomplete lineage sorting, because the lineages of alleles don't get completely sorted as species split apart (<u>Rosenberg and Nordborg 2002</u>).

Fortunately, scientists have several ways to overcome hurdles like these. Rather than studying a single gene, for example, they will routinely sample dozens of them. And when scientists sequence a species' entire genome, they can carry out an even bigger comparison. That's what a team of scientists did in 2012 after sequencing the entire genome of the gorilla (Scalley et al. 2012). They then took the opportunity to address the question of how the great apes (including us) are related to one another. In a number of studies based on smaller amounts of DNA, scientists have generally agreed that chimpanzees and humans are more closely related to each other than either is to the other great apes.

The gorilla genome team compared the genomes of humans, chimpanzees, and orangutans. They found that some segments of human DNA were more similar to those of gorillas than chimpanzees or orangutans. But across the entire genome, they found that only 30 percent of the genes linked gorillas and humans, while the other 70 percent linked humans and chimpanzees. This imbalance is consistent with humans and chimpanzees having evolved from a close common ancestor, along with some incomplete lineage sorting among the genes. And when the scientists lined up all of the protein-coding genes and tallied the similarities at each base, they found more support for this hypothesis. As <u>FIGURE 7.5</u> shows, chimpanzee DNA is more like ours overall than that of other apes.



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A: In 2012, scientists compared the genomes of humans, gorillas, chimpanzees, and orangutans to understand which ape is our closest relative. Different portions of DNA produced different results. The white region shows the proportion of each region of the genome that indicates humans are most closely related to chimpanzees. The blue shows portions of the genome that indicated a different relationship between humans and apes. About 70 percent of genome points to chimpanzees as our closest relative (top right). B: This tree illustrates how a DNA segment could indicate gorillas and chimpanzees are

more closely related to each other than either is to humans. The percentages show the amount of DNA that differs between humans and other apes. This analysis also supports the hypothesis that chimpanzees are our closest relatives. (Data from <u>Scally et al 2012</u>.)

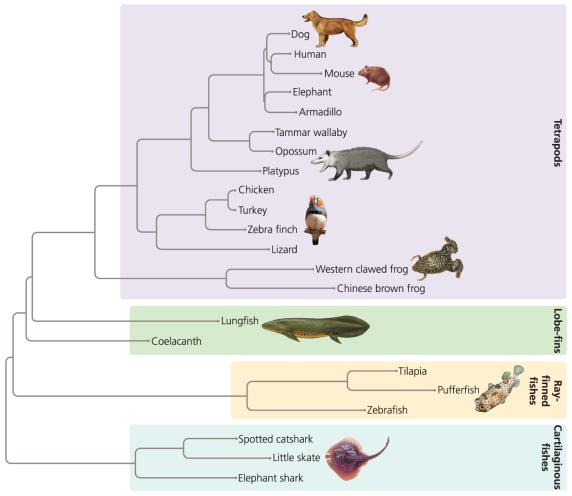
Four Case Studies in Molecular Phylogeny

As scientists overcome challenges like incomplete lineage sorting, they're revolutionizing evolutionary biology. Genomes are historical libraries that have, until relatively recently, been under lock and key. Now that scientists have unlocked them, they can use DNA to test old hypotheses about evolution and develop new ones. Let's take a look at four examples of the insights molecular phylogenies can provide.

The Origin of Tetrapods

As we saw in <u>Chapter 4</u>, scientists who have studied fossils have put forward the hypothesis that the closest living relatives of tetrapods are lobe-finned fishes, which today include only lungfishes and coelacanths. That was a fairly precise prediction, since there are around 30,000 species of fishes alive today, only eight of which are lobe-finned fishes.

More recently, geneticists have sequenced the DNA of tetrapods and fishes and studied it to explore their phylogeny. As DNA-sequencing technology has increased in speed and fallen in price, they've been able to decipher the entire genomes of many of these species. In 2013, an international team of scientists examined 21 different species, comparing 251 of their genes (Amemiya et al. 2013). They reconstructed a phylogeny shown in FIGURE 7.6. Their analysis shows lungfish as the closest relative to tetrapods, followed by coelacanths. Thus, scientists studying one line of evidence—DNA—have confirmed a hypothesis originally developed from another line of evidence—the anatomy of fossil and living species.



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Scientists studying the anatomy of fossil and living species proposed that lobe-fins are the closest living relatives of tetrapods. A phylogeny based on their DNA, shown here, supports this hypothesis. (Information from <u>Amemiya et al. 2013</u>.)

Humans

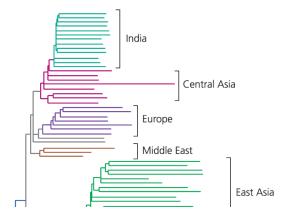
In <u>Chapter 4</u>, we saw how scientists use fossils to learn how humans evolved from extinct hominins. The oldest fossils that show clear signs of belonging to our own species, *Homo sapiens*, came from Africa and date back 200,000 years (<u>page 68</u>). The oldest fossils of our species outside of Africa are some remains in Israel that date to about 100,000 years ago. Younger still are the

first fossils of humans in Europe, Asia, and Australia. In the 1980s, Chris Stringer, a paleo-anthropologist at the Natural History Museum in London, argued that these fossils were evidence that humans arose in Africa and then spread out to the other continents. There, they encountered separate hominin lineages, such as Neanderthals, which later became extinct (Stringer 2012).

According to this scenario, all major ethnic groups of humans—Africans, Europeans, and Asians—descend from African ancestors. Stringer's hypothesis stood in contrast to other models of human evolution in which hominins across the entire Old World were a single species, or a network of closely related species that interbred.

Geneticists have looked to human DNA to test this hypothesis. Sarah Tishkoff is in a particularly good position to do so because she has gathered so much genetic information about people in Africa, where Stringer and others proposed humans originated. Tishkoff and her colleagues analyzed DNA from Africans and compared their genetic sequences with those of people from other parts of the world.

The results of one study are shown in FIGURE 7.7 (Tishkoff et al. 2009). Tishkoff and her colleagues studied 121 African populations, 4 African American populations, and 60 non-African populations. They identified patterns of variation at 1327 sites in their subjects' DNA. Some of these sites, known as nuclear microsatellites, are stretches of repeating DNA that have a very high mutation rate. Tishkoff also looked at loci where DNA had been either inserted or deleted. The tree that best explained the patterns of DNA revealed that most human genetic diversity is in Africa. What's more, all non-Africans form a single clade, suggesting that they descend from a small group of people who left Africa together.





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By comparing DNA from many populations of humans, Sarah Tishkoff and her colleagues produced an evolutionary tree of our species. Because humans have been in Africa much longer than in other parts of the world, Africans today are also much more genetically diverse than other humans. A small group of Africans migrated out of the continent and became the ancestors of today's Europeans, Asians, and people of the New World. As in Figure 7.3, the branch lengths here correspond to the number of mutations accumulated in each lineage. Because many of these mutations are neutral, a

long branch does not mean that a group of people are more "evolved" than others. (Information from <u>Tishkoff et al. 2009.</u>)

Tishkoff's results support the work of other researchers who have studied other genes. It appears that our species first evolved in Africa. Thousands of generations passed before some humans left the continent. Tishkoff's research even offers hints about where in Africa humans originated. She finds the greatest level of diversity and the deepest branches among the people of East Africa—the same region where the oldest fossils of humans have been found. In Chapter 14 we'll take a look at other work by Tishkoff and other scientists that suggests that early humans interbred with Neanderthals and other extinct hominins.

Darwin's Finches

As we saw in <u>Chapter 2</u>, Darwin was struck by the differences among the finches of the Galápagos Islands—so much so that they helped him come up with his theory of evolution. There are finches that nest in cactus, sleep in cactus, mate in cactus, drink cactus nectar, and eat the flowers, pollen, and seeds of cactus. There are two species of finches that use tools: they pick up a twig or a cactus spine, trim it to shape with their beaks, and then poke it into bark on dead branches to pry out larvae. There are finches that eat green leaves, which is practically unheard of for birds to do. Still other finches perch on the backs of Nazca boobies and peck at their wings and tails, drawing blood, which they then drink. There are finches that ride on the backs of iguanas and eat their ticks.

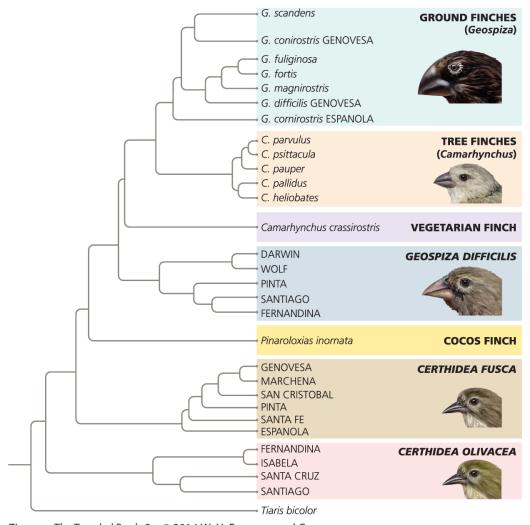
As we saw in the last chapter, Peter and Rosemary Grant have documented changes in the beaks of Darwin's finches through natural selection in as little as a few years. They are also intrigued by the evolution of Darwin's finches over the course of millions of years. They'd like to know where the birds came from and how they evolved into their current diversity.

Of all Darwin's finches, only one does not live on the Galápagos Islands: the Cocos finch, which lives 800 kilometers (500 miles) away on Isla del Coco, another remote island in the east Pacific. No species of Darwin's finches lives on the mainland of South America. A few potential explanations

could account for this arrangement of finches. Perhaps the Galápagos Islands were colonized by several different finch species, each of which gave rise to new species on the islands. Or perhaps some Cocos finches that ended up on the Galápagos Islands gave rise to the many species of Darwin's finches.

The Grants can't turn to the fossil record for answers. The fragility of the birds' skeletons and the harsh climate of the Galápagos Islands means there are no fossils of the birds to examine. So the Grants and their colleagues have been reconstructing the evolutionary history of Darwin's finches by using DNA. The researchers collect blood samples from the birds and then compare genetic sequences from one species of Darwin's finch to another as well as to other species of birds that have been suggested as close relatives.

The Grants and their colleagues found that Darwin's finches are related to each other as illustrated in FIGURE 7.8 (Sato et al. 2001). Darwin's finches are more closely related to each other than they are to any other known bird, suggesting that they did not evolve independently from different ancestral species. The tree also shows that Cocos finches share a closer ancestry with some species of Darwin's finches than they do with others. It's therefore unlikely that Darwin's finches originated from Isla del Coco. Rather, Cocos finches most likely evolved from Darwin's finches that migrated from the Galápagos Islands to Isla del Coco.



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The DNA of Darwin's finches records their evolution from a single common ancestral population. The position of the Cocos finch in this tree (yellow bar) indicates that it evolved from ancestors already present in the Galápagos. (Information from <u>Grant and Grant 2002</u>.)

As for the ultimate origin of Darwin's finches, the DNA study linked Darwin's finches to a group of birds known as seed-eating tanagers that live in South America, Central America, and the Caribbean. It's not yet clear which species of that group is the closest relative of Darwin's finches, but some scientists argue that the ancestors of the dull-colored grassquit of Ecuador gave rise to them (Sato et al. 2001).

How did seed-eating tanagers end up thousands of kilometers away from the Amazon, on lonely islands in the Pacific? One possibility is that a flock of birds were flying over a volcano in the Andes when it erupted. Trying to escape the deadly cloud of ash, they headed out over the ocean, where stormy winds swept them far out to sea. The Galápagos Islands saved them from death. Once they settled on the islands, the birds evolved into the many species alive today—and perhaps others that have since become extinct (Grant and Grant 2008).

Human Immunodeficiency Virus

Molecular phylogeny has become important in the search for the origins of diseases. One of the world's worst pandemics in modern history is caused by HIV. In 2011, an estimated 34 million people worldwide had HIV infections, and an estimated 1.7 million people were dying of AIDS-related causes every year. Given the global suffering that HIV causes today, it may come as a surprise to learn that HIV is a latecomer. Scientists first became aware of it in the early 1980s among young men in California. Soon after, it became clear that HIV was a global epidemic, spread by sexual transmission and contaminated needles.

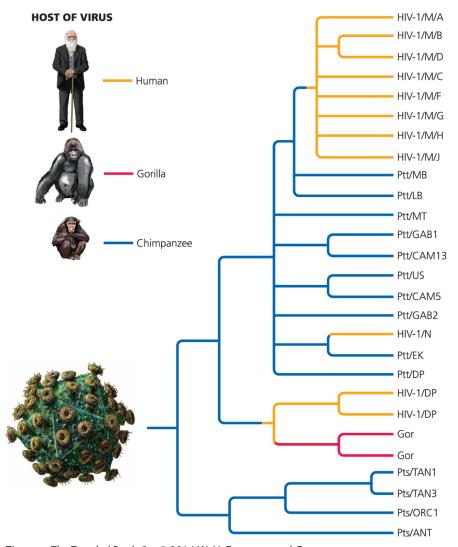
Scientists have searched through medical records and blood samples for earlier cases of HIV infection that might have been overlooked. The earliest known specimen of HIV comes from a blood sample taken from a patient in 1959 in Kinshasa, the capital of the Democratic Republic of Congo.

The mysterious appearance of HIV led to many speculations about where it came from—including accusations that vaccinations contaminated by a monkey virus introduced it into people. But when scientists reconstructed the evolutionary tree of the virus and its relatives, they rejected those claims.

As soon as scientists discovered HIV, it was clear that it belonged to a group known as the lentiviruses. Lentiviruses infect mammals, such as cats, horses, and primates, typically by invading certain types of white blood cells. Genetic studies revealed that HIV is most closely related to strains of lentivirus that infect monkeys and apes—collectively, these strains are known as simian immunodeficiency virus, or SIV for short. These studies have also revealed that what we call HIV is actually several separate

lineages of viruses, each of which evolved from SIV independently (<u>Van</u> <u>Heuverswyn et al. 2007</u>).

The virus known as HIV-1, which causes the vast majority of AIDS cases, is most closely related to the SIV viruses that infect chimpanzees. HIV-2 belongs to a group of SIV viruses that infect a monkey known as the sooty mangabey. A closer look at HIV-1 (FIGURE 7.9) reveals that it initially evolved in a subspecies of chimpanzee, *Pan troglodytes troglodytes*, found in Central Africa (Sharp et al. 2011). Today, HIV-1 is composed of three distinct lineages, each apparently the result of a separate jump from chimps to humans. SIV most likely evolved into HIV-1 as hunters killed apes to sell in a growing "bushmeat" trade. Viruses in the blood of the primates could have entered cuts in the skin of the hunters. Once inside, the virus populations would have begun to adapt to their new human host.



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The evolutionary tree of HIV-1 reveals how the virus hopped from chimpanzee hosts to humans three times. (Information from <u>Sharp and Hahn 2011</u>.)

Knowing the structure of the HIV tree allows scientists to pinpoint the specific adaptations that may have allowed HIV to infect humans. As all three lineages of HIV-1 evolved from chimp-virus ancestors, they acquired the same mutation encoding the same new amino acid in the same position in the same protein (Wain et al. 2007). No SIV virus found in chimpanzees codes for that amino acid in that position, and the phylogeny suggests that it

arose independently each time the virus adapted to human hosts. This mutation altered a gene encoding the shell of the virus, and experiments suggest that it was crucial to the success of the new HIV viruses in humans. It's possible that the mutation allowed the virus to better manipulate its new hosts into building new copies of itself.

Studies like these allow scientists to better understand the evolution of human disease and may help better predict the emergence of new pathogens —a subject we'll explore in Chapter 15.

Natural Selection versus Neutral Evolution

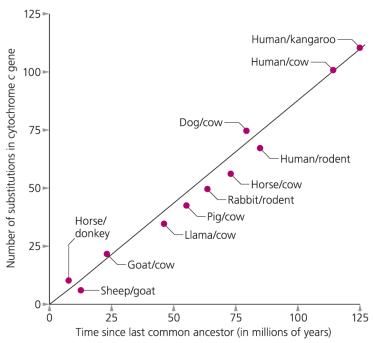
Scientists use molecular phylogenies to solve mysteries about particular groups of species, but they also use them to tackle broader questions about evolution in general. One of the biggest questions is how much of life's diversity can be explained by selection and how much by other evolutionary mechanisms.

Evolutionary biologists agree that natural selection is crucial to the evolution of complex morphology and behavior. Because it occurs in response to an organism's environment, it can produce adaptations that enable it to thrive in that environment. It's also true, as we saw in Chapter 6, that genetic drift can drive neutral mutations to fixation. As we saw in Chapter 5, many non-protein-coding regions of the genome have no apparent function. Even protein-coding genes can pick up mutations that have no effect on their fitness. Each amino acid in a protein is encoded in a three-nucleotide codon. If a mutation switches a codon to another one that encodes the same amino acid, the protein will remain the same. Scientists call this type of mutation a synonymous substitution.

In 1968 Motoo Kimura, a biologist at the Japanese National Institute for Genetics, developed a mathematical theory to explain the role of such neutral mutations in evolution. According to his theory, much of the variation in genomes was the result of genetic drift (Kimura 1968, 1983). As Kimura and other researchers developed this theory, they predicted that neutral mutations become fixed in populations at a roughly regular rate. When a population split into two lineages, each lineage would acquire its own unique set of neutral mutations. The more time that passed after the lineages diverged, the more different mutations would be fixed in each one.

When scientists began comparing proteins and genes from different species, they found compelling support for Kimura's theory. Walter Fitch of the University of Wisconsin and Charles Langley of the National Institute of Environmental Health Sciences in North Carolina, for example, studied a

gene for a protein called cytochrome c in 17 species, including humans and horses (<u>Langley and Fitch 1974</u>). The genes were nearly identical in each species, except for a few mutations. They worked out how many mutations had accumulated in each lineage after they diverged from a common ancestor. Fitch and Langley then asked paleontologists to estimate when those lineages had split based on the fossil record. The more distantly related two species were, the more mutations had accumulated in each lineage since they split from their common ancestor (<u>FIGURE 7.10</u>). What made the graph especially striking was the clocklike regularity by which the mutations became fixed.



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FIGURE 7.10

Mutations become fixed at a roughly clocklike rate. This graph shows how distantly related species, such as humans and kangaroos, have many more differences in the cytochrome c gene than do closely related species like sheep and goats. (Data from Moore and Moore 2006.)

The Molecular Clock

As predicted by the neutral theory of evolution, mutations accumulate at a roughly clocklike rate over millions of years. Once scientists realized this, they realized something else: they could use mutations to tell time. By counting the number of mutations in a species' cytochrome c gene, for example, they could estimate how long ago its ancestors branched off from our own. This method of telling time has come to be known as the molecular clock.

To use the molecular clock, scientists must first calibrate it. Different stretches of DNA mutate at different rates. Sites in genes that encode proteins accumulate mutations at a slow rate. That's because mutations to those sites can alter proteins—which can lead to a loss of fitness. Pseudogenes, by contrast, acquire mutations 10 times faster, because they no longer encode proteins and so most mutations to them will be neutral. To measure the divergence of species separated by hundreds of millions of years, a slow-evolving segment of DNA will be more accurate than a fast-evolving one. Scientists have also found that the molecular clock can run slower or faster in different lineages, its speed influenced by factors like the size of populations and how long organisms take to mature and reproduce.

Scientists have developed statistical methods to address these challenges, and as a result they've been able to use the molecular clock to investigate many questions about the history of life. As we saw earlier, the Grants used molecular phylogenetics to determine that Darwin's finches all descend from a single founding event. To find out when that event occurred, they turned to the molecular clock. The Grants and their colleagues found that all 13 species can be traced back to a common ancestor that lived between 2 and 3 million years ago.

To explore the significance of this result, the Grants turned to the research of geologists who study the islands. The Galápagos Islands emerged as the crust of the Pacific seafloor moved over an underlying blob of hot rock. The blob pushed up the crust, forming a volcanic island. Gradually, the crust moved away and the island cooled and sank, while a new island formed

nearby. Geologists can determine the age of these islands by examining the radioactive isotopes in their rocks (see <u>Chapter 3</u> for an explanation of radiometric dating). They've found that the first Galápagos Islands formed some 10 million years ago and then later were submerged. A map of the Galápagos Islands from 2 to 3 million years ago, when the ancestors of Darwin's finches arrived, would have looked radically different than a map from today. Instead of 18 islands as there are today, there were perhaps only five (<u>Grant and Grant 2008</u>).

After the ancestors of Darwin's finches arrived, new islands continued to form as older islands sank back into the sea. The climate was changing as well. Starting 2.75 million years ago, the planet cooled. It also started to swing through cycles, as periodic ice ages spread glaciers across much of the Northern Hemisphere. Sea level rose and fell, shrinking and then expanding the size of the islands. The plants on the islands responded to the changing climate as well, giving rise to new ecosystems. This combination of geological, climatic, and ecological changes drove the remarkable burst of evolution that so impressed Darwin.

An unexpected benefit of the molecular clock has been its usefulness in tracking the origins of diseases. Viruses replicate at an extraordinary rate—a single virus can produce a billion progeny in a day—and they lack repair enzymes to fix errors in new copies of their genes. As a result, their mutation rate is far higher than that of animals. Moreover, their molecular clock spins far faster, making it possible to use it to explore the history of viral outbreaks that occurred within the past century.

Once scientists determined that HIV had evolved from viruses that infect chimpanzees, for example, they used the molecular clock to estimate when that shift took place. Researchers based at Los Alamos National Laboratory compared the RNA from 159 HIV-1 viruses that had been isolated from blood samples collected from patients at different times during the previous two decades. They calculated the most likely rate at which the virus genes mutated, based on how different the viruses were from one another and how old they were.

Rather than requiring that the viruses all follow one clock strictly, they allowed the mutation rate to vary from branch to branch, and even from site to site within the genes. The scientists estimated from the isolates collected during the 1980s and 1990s that the common ancestor of HIV-1 existed

sometime between 1915 and 1941. The most likely year was 1931 (Korber et al. 2000).

Scientists got an unexpected chance to test this estimate when a sample of HIV turned up in a tissue sample dating back to 1959. Because they hadn't used the old viruses in their initial estimate, this sample could serve as a check on the accuracy of their molecular clock. The 1959 virus was decades older than the ones the Los Alamos team had analyzed, so the branch that joined it to the common ancestor of all HIV-1 was shorter. And that meant that it should have fewer mutations than younger viruses. That indeed is what the scientists found. In fact, the number of mutations in the 1959 virus was close to that predicted by the earlier study (Worobey et al. 2008). The old HIV sample thus supported the conclusions from the molecular phylogeny of HIV described earlier in this chapter. Rather than being accidentally introduced in vaccines in the 1950s, the virus likely had evolved decades earlier from viruses that infect chimpanzees.

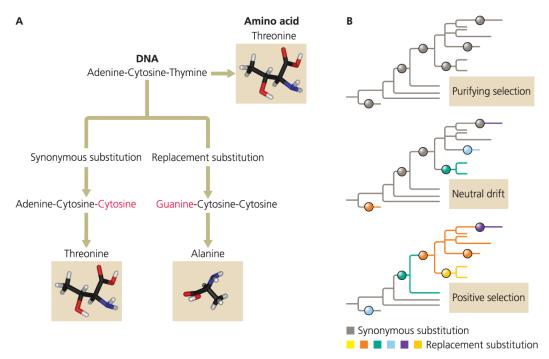
Footprints of Selection

In <u>Chapter 6</u>, we looked at a number of ways scientists study natural selection. They can rear bacteria in their labs and measure their fitness. They can track populations of birds from year to year through droughts and floods. They can even look back over thousands of years by examining DNA for signs of recent selective sweeps. But these methods falter when we look back tens of thousands of years ago, and they collapse completely when thousands of years become millions. Fortunately, molecular phylogenies give scientists an opportunity to reach back to these ancient episodes of evolution.

This time travel can be carried out in a number of ways, but they generally rely on the same basic approach. Scientists compare different versions of the same gene in a number of individuals—either within a single species, or across a number of species—and run tests to see if the differences between them can be explained by neutral evolution. If scientists can reject that possibility, they can conclude that natural selection is at work. (This method is similar to the way scientists used the Hardy–Weinberg model to test for evolution—see page 127.)

One of these methods takes advantage of the fact that mutations to the same gene can have different effects on a phenotype. To understand how this method works, we have to consider how cells read out DNA to express proteins. As we saw in Chapter 5, genes encode each amino acid in a protein with three bases—a codon. But codons do not correspond to amino acids in a neat, one-to-one fashion. A single amino acid may actually correspond to more than one codon.

For example, a codon with the sequence adenine-cytosine-thymine corresponds to the amino acid threonine. But so does the codon adenine-cytosine-cytosine. Thus, a mutation that converts the third position from thymine to cytosine will have no effect on the resulting protein (FIGURE 7.11A).



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Positive natural selection can lead to an unusually large number of mutations that change the structure of proteins, called replacement substitutions. A: Synonymous substitutions alter a codon without changing the amino acid it encodes. Changing thymine to cytosine, as shown here, still encodes the amino acid threonine. The protein is thus unchanged. Changing adenine to guanine, on the other hand, changes the amino acid altogether. B: Scientists can look for evidence of selection by comparing substitutions of different genes. *Top*: Replacement substitutions in this gene lower the fitness of organisms, so they tend to disappear from populations. Synonymous substitutions are thus far more common. *Middle:* Nucleotides in this gene are equally likely to acquire synonymous or replacement substitutions. This is a sign of neutral evolution due to drift. *Bottom:* This gene acquires replacement substitutions that raise fitness. They are favored by natural selection and become fixed at a greater rate than synonymous substitutions.

On the other hand, if the mutation changes the *first* base from adenine to guanine, the amino acid will change from threonine to alanine. The first mutation causes no phenotypic change to the protein, while the second one does. These two kinds of mutations are called, respectively, synonymous substitutions and non-synonymous substitutions.

Both kinds of mutations can become fixed in a population, but different factors may be involved. Synonymous substitutions have no effect on a protein's structure, which means that they can't change an organism's fitness. As a result, the only way they can become fixed is through genetic drift.

A non-synonymous substitution, on the other hand, *does* change the structure of the protein. If that change has no effect on fitness, it can become fixed only by genetic drift. But if it does raise an organism's fitness, selection can fix it. Conversely, a non-synonymous substitution can have a devastating effect on an organism. Negative selection can eliminate a non-synonymous substitution from the population.

Let's now consider a gene that has been evolving for millions of years. New mutations arise in the gene in individuals from time to time at a roughly regular rate. If the gene is experiencing no selection—a pseudogene, for example, that no longer encodes a functional molecule—then the only way for any of those mutations to reach fixation is through genetic drift. Under such conditions, you'd expect that the odds of a non-synonymous substitution becoming fixed would be equal to that of a synonymous one (Bell 2008).

Scientists can calculate these probabilities by reconstructing the evolutionary history of a gene. By comparing variants of the gene in different individuals, they can infer what its sequence was in their common ancestor. They can then track which sites in the gene changed over time through the fixation of mutations. By tallying the number of non-synonymous and synonymous substitutions, they can see how likely it was for each kind to occur. If the ratio between the odds for the two kinds of substitutions is 1, then genetic drift rather than selection likely was responsible for the pattern of mutations we see in the gene.

But if the ratio is far from 1, then we can reject the hypothesis that neutral evolution is responsible for the pattern. We can then explore how different kinds of selection might have produced it instead (FIGURE 7.11B). Let's say that a gene has experienced strong positive selection over millions of years, as mutations alter the protein it encodes. Such a gene would accumulate a high number of non-synonymous substitutions.

Now consider a gene that carries out an essential function that's easily disrupted by mutations to non-synonymous sites. Selection will eliminate those mutations from populations—while ignoring the synonymous

substitutions. Purifying selection, as this process is known, will lead to a gene with a very low number of non-synonymous substitutions compared to synonymous ones (<u>Charlesworth and Charlesworth 2010</u>).

By detecting natural selection in this way, scientists are pinpointing some of the genes that may have helped to make us uniquely human. One of the most tantalizing of these genes is known as *FOXP2*, the first gene ever clearly linked to language. People with mutations to *FOXP2* suffer devastating difficulties in speaking and understanding grammar (Enard et al. 2002).

In most of the mammals that scientists have surveyed, the amino acid sequence of the FOXP2 protein has not changed for tens of millions of years (FIGURE 7.12). The FOXP2 gene carried by a chimpanzee is practically identical to that of a mouse. In humans—and humans alone—two amino acids have changed in the protein in just the past 6 million years. That change represents a powerful episode of natural selection, given how little the gene has changed in other lineages. It's possible that the transformation of FOXP2 helped to give rise to full-blown language in our species (Chapter 14).

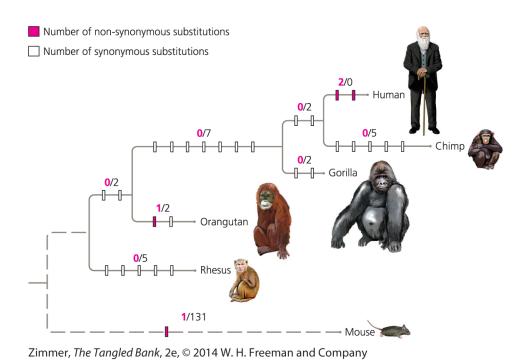


FIGURE 7.12

A gene linked to language, called *FOXP2*, has experienced strong positive selection in the human lineage. In other mammal lineages, *FOXP2* has acquired few non-synonymous substitutions over millions of years. (Data from <u>Enard et al. 2002</u>.)

Deciphering the Genome

Evolutionary trees can shed light not only on protein-coding DNA but also on the 98.8 percent of our DNA that does not encode proteins. Most noncoding DNA has no obvious function, yet scientists have found essential segments sprinkled in these "gene deserts." Finding these functional segments of non-protein-coding DNA is difficult, however, because they are often extremely small—only a few dozen bases long in some cases. To make the challenge even tougher, regulatory regions don't have the nice regularity that the genetic code imposes on protein-coding genes.

One way to find these segments is to take advantage of evolutionary history. When scientists compare the same segment of noncoding DNA in a number of related species, they usually find a number of different mutations—the result of neutral evolution. But sometimes they'll find a segment that has remained almost unchanged in every species. Purifying selection can preserve these so-called conserved regions, if they play some important role.

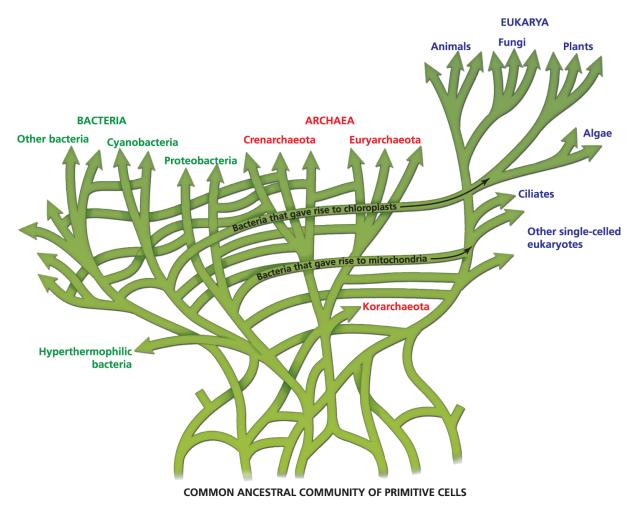
Box 7.1

The Tree of Life—or Perhaps the Web of Life?

Darwin's metaphor of a tree of life goes a long way to explain the patterns we see in nature. But it is not the whole story. The branches of the tree represent the flow of genetic information from parents down to their offspring. Scientists have known for decades, however, that genes can move between the branches. Some species can interbreed and produce viable offspring, for example. Viruses can pick up genes from one host and accidentally insert them in another. Some bacteria build complex, pump-like structures to deliver genes to other bacteria. These transfers can take place between two members of the same species, or, less often, between two members of different species. Sometimes

a microbe can become incorporated into a host cell, and then the two species merge into a single organism.

Long after horizontal gene transfer was discovered, many scientists considered it unimportant for the large-scale evolution of life. That has changed (Syvanen 2012). Scientists now know that the genomes of many species are mosaics, assembled from chunks of DNA imported over billions of years from a vast number of different species. To represent this rampant horizontal gene transfer, many scientists are reconceptualizing the tree of life as a "web" of life.



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FIGURE 1 To represent the flow of genes between species, we can draw the tree of life as a web.

In some cases, a region of non-protein-coding DNA is conserved in many species, but altered in one. Such is the case with 202 regions that are conserved in all vertebrates except humans. These segments may have played a crucial role in making us uniquely human. Out of all these 202 segments, the one that has changed the most is called *HAR1*. It turns out to be an RNA-encoding gene that is expressed only in the brain during the development of embryos. Part of what makes us human lies in the unique anatomy of our brain. The discovery of *HAR1* could help us understand the nature of our uniqueness (Pollard et al. 2006).

Sometimes genome comparisons can reveal genes that previously went undetected. Ed Rubin of Lawrence Berkeley National Laboratory in California and his colleagues used this method in a study of genes that control the levels of molecules known as lipids in the blood (<u>Pennacchio et al. 2001</u>). Lipids are an essential part of our cell membranes, but people with high lipid concentrations in their blood face a risk of heart disease.

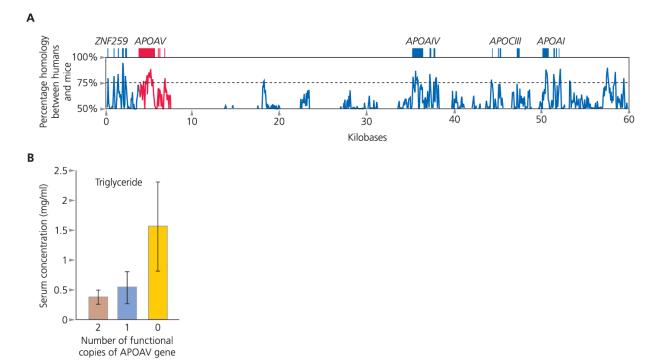
To find genes involved in lipid levels, scientists have used quantitative trait loci (QTL) analysis (page 120) to survey the DNA of people who produce very high or very low levels of the molecules. They discovered a cluster of genes sitting close by each other, called *APOAI*, *APOCIII*, and *APOAIV*. It turns out that mice carry similar versions of the same three genes in the same location in their genome. This striking similarity indicates that this cluster of lipid genes was present in the common ancestor of mice and humans 100 million years ago.

Rubin wondered if these studies had overlooked additional genes involved in controlling lipids. To find them, he and his colleagues searched the 200,000 bases of noncoding DNA surrounding the gene cluster. They lined up the human version of this region with the one from the mouse genome. Different nucleotides were sprinkled along each segment, the result of neutral evolution acting on noncoding DNA. But when they reached a spot 30,000 bases away from the gene cluster, Rubin and his colleagues discovered something odd: a stretch of 1107 bases that were nearly identical in human and mouse DNA.

The scientists hypothesized that these 1107 bases were, in fact, a conserved gene. To see if it was, they looked at its sequence and inferred what kind of protein it would encode. They concluded that the protein would have a series of spirals that are a hallmark of all known lipid-binding

proteins. That was a promising sign, so they began to run experiments to test the function of the gene, which they called *APOAV*. They engineered mice with extra copies of the gene and then analyzed their blood. The scientists found that the extra *APOAV* genes drove down the lipid levels dramatically. Rubin and his colleagues then ran another experiment on another set of mice. In these animals they disabled *APOAV*, so that the mice could not produce the protein. The mice developed much higher lipid levels—supporting the hypothesis that the gene encodes a protein that keeps lipid levels down.

Finally, Rubin and his colleagues turned to humans. They looked at the DNA from a group of people and discovered that they had slightly different versions of the *APOAV* sequence. If the gene controlled lipids in humans, the scientists reasoned, then they might be able to find differences in the health of people who have different versions of *APOAV*. And, just as they had hoped, they discovered that variations in *APOAV* are associated with variations in heart disease.



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FIGURE 7.13

A: Ed Rubin of Lawrence Berkeley Laboratory and his colleagues compared the DNA surrounding three known lipid-binding protein genes (APOAI, APOCIII, and APOAIV) in

humans and mice. The graph shows the level of homology in different regions of DNA in the two species. Rubin and his colleagues then looked at the long stretches of noncoding DNA flanking these genes and other known genes in their neighborhood. Nestled between *APOAIV* and *ZNF259*, an unrelated gene, they found a stretch of DNA with a high level of homology to the lipid-binding protein genes. When they investigated this stretch of DNA, they found a new gene for another lipid-binding protein, which they dubbed *APOAV*. B: The scientists bred mice that lacked one or two copies of *APOAV*. Without the newly discovered gene, mice produced four times more triglycerides, which are fat molecules associated with heart disease. Thus evolution guided the scientists to the discovery of a medically important gene. (Data from <u>Pennacchio et al. 2001</u>.)

By looking back over 100 million years of evolutionary history, Rubin and his colleagues discovered a gene that may play an important role in heart disease. In <u>Chapter 15</u>, we'll look in more detail at how evolutionary biology sheds light on diseases.

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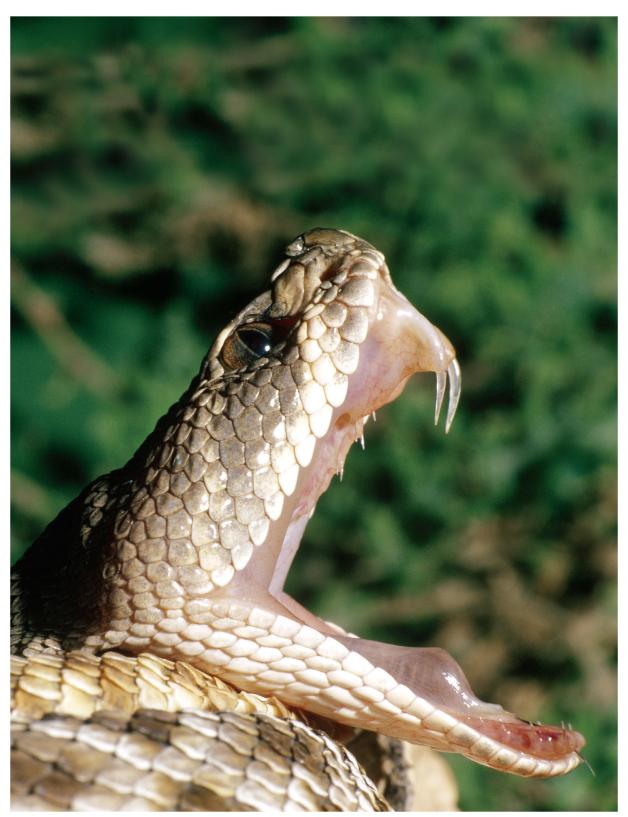
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Adaptation

The Birth of the New



Tom McHugh/Science Source.

Snakes have evolved a complex system of venom molecules, specialized glands, and fangs.

To study evolution, Bryan Fry puts his life on the line. Fry, a biologist at the University of Melbourne, studies the evolution of snake venom, and that means he has to make intimate contact with some of the most lethal animals in the world, from deadly sea snakes to king cobras. Fry is an admitted adrenaline junkie, but he's not reckless. He prepares himself for every encounter so that he comes home safe and sound. It helps to understand snake behavior. Fry knows, for example, that a king cobra signals its dominance over other king cobras by extending up and touching the tops of their heads. To trap a king cobra, Fry first shows it who's boss by tapping it on the head. The cobra bows down in submission, and Fry takes the opportunity to slip it into a bag.



Bryan Fry.

Bryan Fry investigates the evolution of this complicated adaptation by analyzing the genes for venom.

Once back in his lab at the University of Melbourne, Fry can get a close look at the adaptations that make the cobra so fearsome. When a cobra attacks, it bares two long, hollow fangs that it sinks into its victim. Into the wound, the snake delivers venom, produced in glands in the back of its mouth and then delivered through tubes into the hollow fangs. Each species of venomous snake produces a cocktail of venoms, each one exquisitely adapted for disabling the snake's prey. Some snakes make venom that relaxes the walls of the aorta, dropping the blood pressure in a snake's victim until it blacks out. Other venoms lock onto receptors in neurons, causing paralysis. Others interfere with the biochemistry inside muscle cells, causing them to break down rapidly.

In <u>Chapter 6</u>, we saw how natural selection drives alleles to fixation, and how it favors organisms with certain traits over others. But how did these traits emerge in the first place? The snake venom-delivery system did not exist a billion years ago. At some point, it evolved in the ancestors of today's venomous snakes. But a single mutation could not produce all the traits that make a cobra such a deadly animal. The venom-delivery system is made up of proteins, cells, glands, and fangs. How did all the genes required to make something so complex evolve?

That's the question Fry risks his life to answer. It's the same question other biologists are asking about other complex adaptations, such as eyes and limbs. In this chapter, we'll explore their research into the origin of complex adaptations. We'll see how natural selection is essential to their evolution, but we'll also see how their history is far more than the spread of a single allele through a single population. It's an intricate epic of new genes

evolving new functions while old genes take on new jobs. We'll see how scientists investigate the origin of adaptations with many lines of evidence—from fossils to observations of living species to experiments that reveal the functions of their genes.

Innovation in Our Own Time

Complex traits do not arise out of the blue. They arise from older traits, which are modified to take on new functions. The evograms in Chapter 4 offer several examples of this process from the fossil record. The bones of the middle ear in mammals started out as bones in the lower jaw, for example. In 1982, Yale paleontologist Elizabeth Vrba and Harvard evolutionary biologist Stephen Jay Gould dubbed these co-opted elements "exaptations" (Gould and Vrba 1982). They came up with the concept of exaptations long before scientists gained a good understanding of the molecular evolution of complex traits. Remarkably, exaptation turns out to be an important process on the molecular level as well.

A striking example of molecular exaptation recently emerged in the laboratory of Richard Lenski. In <u>Chapter 6</u>, we learned about Lenski's 25-year experiment with evolving bacteria. At the beginning of his experiment, he set up 12 genetically identical *E. coli* lines and fed them all a meager diet of glucose. As the lines reproduced, they mutated and adapted to their new conditions. But in one of those lines, something remarkable happened: a new trait evolved. The bacteria acquired the ability to feed on a molecule that was off-limits to the other microbes.

The first sign of this remarkable event was in 2003, when Lenski and his students noticed something odd. Overnight, one of the 12 flasks had become much cloudier than the others.

In a microbiology lab, cloudiness is a sure-fire sign that the bacteria in a flask have experienced a population explosion. At first the team suspected that some other species of bacteria had slipped into the flask and were overgrowing the native *E. coli*. But they found that the bacteria making the medium cloudy was their own *E. coli*—the descendants of the original ancestor that Lenski had used to start the entire experiment. Somehow the bacteria in this one flask had evolved a way to grow more than the bacteria in the other 11.

The scientists studied the bacteria and discovered they had evolved a new trait—the ability to feed on an ingredient in their medium that the other E.

coli couldn't. Their new source of food, a compound called citrate, is an ingredient in the broth in which the bacteria grow. Citrate, the compound that makes lemons tangy, creates the best conditions in the medium for *E. coli* to take up iron.

Scientists sometimes find natural strains of E. coli that can feed on citrate in the presence of oxygen. They acquired this ability through horizontal gene transfer, taking in plasmids from other species (page 114). Most other strains of E. coli, on the other hand, can feed on citrate only in the absence of oxygen. It's a strategy that they rely on when they can't use aerobic metabolism to grow on higher-quality sources of energy, such as glucose.

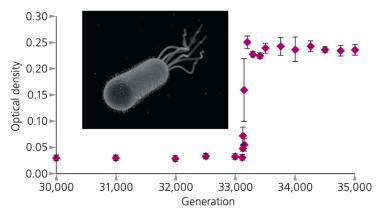
When *E. coli* senses that it is in an oxygen-free environment with citrate, it responds by expressing a set of genes known collectively as *citT*. The genes encode a protein that ends up in the membrane of the microbe. There, it pumps out a compound called succinate while pumping in citrate. *E. coli* can then harvest the energy in the citrate to power its metabolism. If the oxygen level in its environment rises again, *E. coli* silences the *citT* genes and stops feeding on citrate.

E. coli's inability to grow on citrate in the presence of oxygen is a hallmark of the species. When microbiologists are trying to determine the species of bacteria causing an infection, for example, they will culture the microbes on a petri dish that contains only citrate. If bacteria start to grow on it, they know that they're dealing with a species other than *E. coli*.

It was thus a surprise to Lenski and his students to find that bacteria that they *knew* were *E. coli* were feeding on citrate. The bacteria could not have acquired this trait through horizontal gene transfer because Lenski was careful to start the experiment with a strain of *E. coli* that lacks the ability to acquire genes from other species. Instead, the citrate feeding must have evolved after Lenski started his experiment. This was not simply a case of natural selection enabling a species to do something better. This was a case of doing something new.

Christina Borland, a postdoctoral researcher in Lenski's lab, began the investigation of this strange evolution, which was later taken over by Zachary Blount—then a graduate student in Lenski's lab and now a postdoctoral researcher there (Blount et al. 2012). Borland stopped feeding the citrate-feeding bacteria their regular diet of glucose. The bacteria kept growing, she found, demonstrating that they could now thrive on citrate alone.

Borland then defrosted the bacteria's ancestors and fed them citrate to figure out at what stage in their evolution the citrate feeding evolved. In the bacteria from around generation 31,000, she found, only a tiny fraction had the ability to grow on citrate, and they did so very slowly. Over the next few thousand generations, these bad citrate feeders got better—so much better that they were able to grow faster than bacteria that couldn't feed on citrate. They then swiftly came to dominate the population in the flask (FIGURE 8.2).



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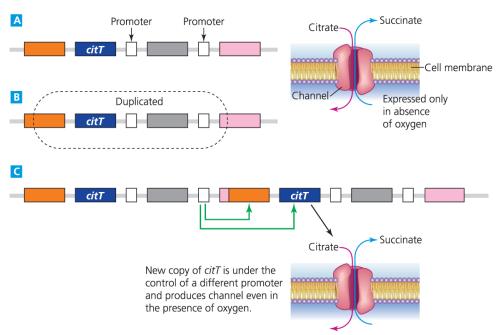
FIGURE 8.2

As Richard Lenski's bacteria adapted to the laboratory environment, one lineage evolved a new trait: the ability to feed on citrate. As the bacteria evolved to eat more citrate, they grew to greater numbers and made their flask cloudier. "Optical density" on this graph is a measure of this cloudiness. (Data from Blount et al. 2008.)

Blount's later research revealed that the evolution of this new trait took place in three stages, like chapters in a story. In the first chapter, the bacteria acquired mutations that would open the way for feeding on citrate. In the second chapter, the bacteria actually became able to feed on citrate. And in the third chapter, they became much better at doing so. It usually makes sense to start a story with Chapter One. But in this case, it's better to start with Chapter Two: The Birth of the Citrate Feeders.

Blount and his colleagues sequenced the genomes of some of the *E. coli* and found some of the genetic changes that made it possible for them to feed on citrate in the presence of oxygen. One of the most important of these

mutations was a duplication (FIGURE 8.3). A 2933-base-pair-long segment of DNA that included citT was copied and then inserted next to the original one. The duplicated segment also included part of an adjacent gene called rnk, as well as the promoter sequence that controls when rnk is turned on.



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FIGURE 8.3

A key step in the evolution of citrate feeding in *E. coli* was an accidental duplication of a segment of its DNA. A: The segment contained a gene called *citT*. It encodes a transporter protein that draws citrate into the cell by exchanging succinate. The gene is expressed only in the absence of oxygen. B: This figure shows the region that was duplicated. C: The new copy of the DNA segment was inserted next to the old one. Its new copy of *citT* now came under the control of a promoter that expressed the gene in the presence of oxygen. As a result, the bacteria could now feed on the citrate in the laboratory flask. (Information from <u>Blount et al. 2012</u>.)

Thanks to this accident, the new copy of citT came under the control of rnk's promoter. And that promoter is switched on in the presence of oxygen, not in the absence of it.

When this mutation occurred around generation 31,500, it allowed only some *E. coli* to grow slowly on the citrate in its flask. At first, the citrate

merely supplemented the regular diet of glucose. For a time, these slow citrate feeders were in the minority among regular glucose feeders. But then, in Chapter Three, life got better for these feeble citrate eaters. More mutations led to further duplications of *citT*, along with its oxygen-sensitive *rnk* promoter.

To judge the effect of these additional duplications, Blount ran an experiment. From generation 32,000, he thawed bacteria that did not carry the mutations allowing them to feed on citrate. Into these bacteria, he inserted many copies of the duplicated citT genes and their new promoter. Immediately, the bacteria were able to grow rapidly on citrate.

Along with the extra copies of citT, the bacteria acquired other mutations that likely improved their ability to break down citrate. Lenski and his colleagues are now testing these mutations to see how they've improved E. coli.

The most intriguing—and still mysterious—chapter of the history, however, is Chapter One: what happened before the gene duplication that allowed the bacteria to start feeding on citrate? Blount and his colleagues have carefully studied the evolution of the bacteria in the generations leading up to the emergence of citrate feeding. They've found evidence that the evolution of citrate feeding could occur only after earlier mutations had opened the door.

This evidence first came to light when Blount decided to thaw out early stages in the citrate-feeding line and see if those bacteria could also evolve the ability. Was this new adaptation the result of a mutation that would arise sooner or later? Or was it contingent on a specific sequence of many mutations happening to strike a lineage of bacteria?

To find out, Blount went back in time again. The researchers thawed out bacteria from different points in the history of the lineage and started 72 new populations. Each population was allowed to evolve for 3700 generations in the same low-glucose medium where citrate feeding had evolved.

In some of the trials, the bacteria evolved into citrate eaters once more—but only if they came from after generation 20,000. If the bacteria came from earlier than generation 20,000, they never acquired this exceptional ability. This discovery suggested that by generation 20,000, the bacteria had acquired mutations that were essential for evolving into citrate feeders.

Blount tested this idea with a series of new experiments. In one of these experiments, he thawed out the original ancestor of a line. Into those early bacteria, he injected copies of the *citT* genes. Unlike the result in his earlier transplant experiment, the ancestral bacteria could not start feeding on citrate. They apparently were missing the necessary mutations to take advantage of extra copies of *citT*. But bacteria from after generation 20,000 could benefit from the transplant.

It's not clear yet what effects these "preparatory" mutations had on the bacteria. Blount and his colleagues suspect that they might have provided some immediate benefit for feeding on glucose and thus spread through the population. But those mutations may have had hidden side effects—they may have opened the way for later generations of bacteria to start feeding on citrate.

Ancient Borrowings

Lenski's bacteria are relatively simple organisms. Animals are far more complex—we humans develop from a single fertilized cell, which gives rise to a body made up of 10 trillion cells divided into hundreds of different cell types. Yet the cells in our brains contain the same set of genes as the brains in our muscles or our stomachs. To understand how this complexity has evolved, we can get some guidance from humble *E. coli*. In both animals and microbes, for example, genes can take on new functions through duplications and changes to their control regions (**FIGURE 8.4**). One of the most striking illustrations of these processes is the evolution of snake venom (<u>Casewell et al. 2013</u>).

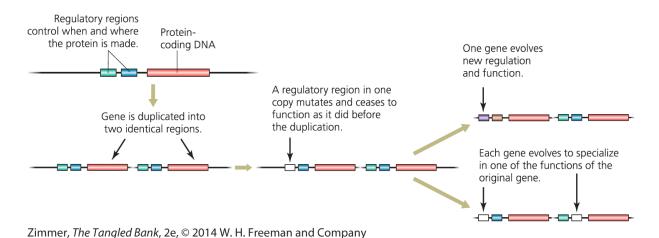


FIGURE 8.4

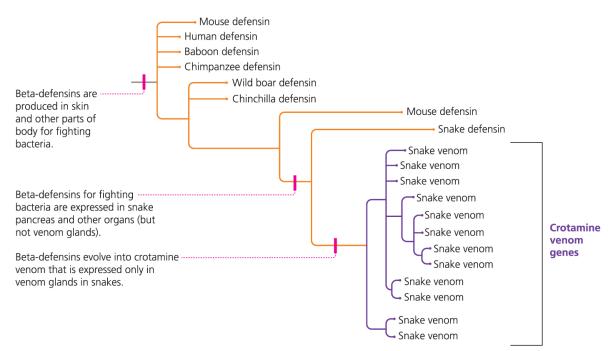
When genes duplicate, one copy can take on a new function. It can evolve through mutations to the coding region that change the structure of its protein. Or the mutations can alter segments of DNA that control when and where the gene is expressed.

Bryan Fry and his colleagues illuminated this evolution by isolating genes for venom from a wide range of snakes. They collected cells from the venom glands of snakes and determined which of their genes were active. About half of the active genes turned out to be ordinary housekeeping genes that carry

out basic processes in all cells. The rest of the active genes encoded venoms. Fry and his colleagues sequenced these venom genes and compared them to figure out how they evolved from an ancestral gene.

Each species produced its own distinctive cocktail of venoms, but each venom gene typically showed a close kinship with venom genes in closely related snakes. The pattern suggests that venomous snakes inherited genes for venom from a common ancestor. After their lineages diverged, the venom genes were shaped differently by natural selection.

Fry and his colleagues reconstructed the history of these venom genes with evolutionary trees. FIGURE 8.5 shows one of these trees, which illustrates the evolution of a muscle-destroying venom called crotamine. Fry compared crotamine genes with one another as well as with other genes in snakes and other vertebrates. The tree reveals that the closest relatives of crotamine genes are defensin genes, which are expressed in the pancreas of snakes. Snakes use defensins to fight infections. Pigs, mice, and humans make defensins as well.



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FIGURE 8.5

Bryan Fry and his colleagues sequenced genes for a venom called crotamine in snakes and compared those genes to related genes in other animals. This evolutionary tree

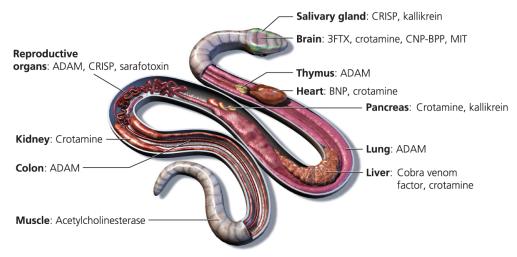
shows how crotamine genes are closely related to beta-defensins, bacteria-fighting molecules found in many vertebrates. (Information from <u>Fry et al. 2006</u>.)

These results support the hypothesis that the defensins originally evolved in a common ancestor of snakes and mammals some 300 million years ago. As new lineages of animals split off from that ancestor, they inherited that ancestral defensin gene. The gene underwent many rounds of duplications, producing a family of similar genes. Some of these genes evolved to attack pathogens in the pancreas.

The ancestors of today's snakes inherited those duplicated genes, one of which experienced a mutation that affected where the protein was made. Instead of being made in the pancreas, it was produced by cells in snake mouths. When the snakes bit their prey, they now released this defensin into the wound.

Fry proposes that further mutations to the duplicated defensin gene changed its protein's shape, so that it began to take on a new function. Instead of fighting pathogens, it began to damage muscles. Further mutations made defensin increasingly deadly, and subsequent gene duplications eventually gave rise to an entire family of these venom genes.

Fry and his colleagues have drawn similar trees for more than two dozen venom genes. Some evolved from genes expressed in the heart, while others were expressed initially in the brain, in white blood cells, and in many other places in the snake body (FIGURE 8.6). An Australian snake known as the inland taipan produces a venom that causes its victims to "black out," for example. The gene for this venom evolved from proteins that slightly relax the muscles around the heart. Once these proteins evolved into venom, this slight relaxation became a rapid drop in blood pressure. In each case, venoms evolved through gene duplication, gene recruitment, and fine-tuning mutations to the genes themselves.



Snake toxin	Effects
3FTx	Neurotoxin
Acetylcholinesterase	Disruption of nerve impulses, causing heart and respiratory failure
ADAM	Tissue decay
BNP	Acute low blood pressure
CNP-BPP	Acute low blood pressure
Cobra venom factor	Anaphylactic shock
CRISP	Paralysis of peripheral smooth muscle, hypothermia
Crotamine	Muscle decay and neurotoxicity
Kallikrein	Acute low blood pressure, shock, destruction of blood-clotting factors
MIT	Constriction of intestinal muscles, resulting in cramping, increased perception of pain
Sarafotoxin	Acute high blood pressure

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FIGURE 8.6

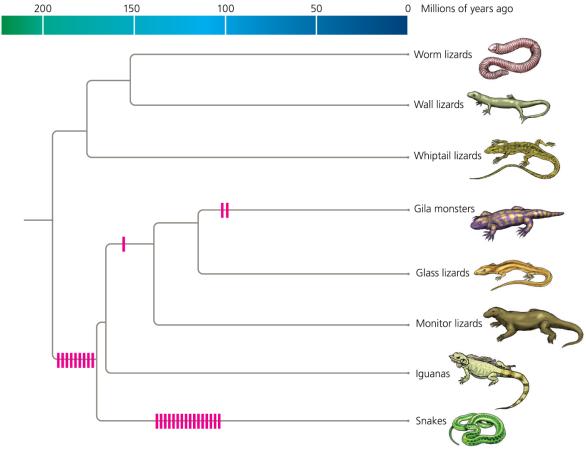
Venom genes have been recruited from genes expressed in many organs of snakes.

The venom-delivery system in a taipan is, of course, more than just the venom genes. It also includes the venom glands and the fangs for delivering the venom deep into the flesh of its prey. It may be hard to imagine how this complex system evolved, since all the parts seem to depend on each other. Yet when Fry looked closer at the venom system, he made a major discovery about how it evolved.

Fry wondered if gene recruitment for venom production took place independently in each lineage of venomous snakes, or if some venom genes evolved before their lineages split and before the evolution of complex venom-delivery systems. He discovered that some venom genes are ancient, having evolved in the common ancestor of all snakes—even snakes not previously recognized as venomous. Garter snakes, for example, lack hollow fangs and high-pressure delivery systems. But Fry discovered that these relatively harmless snakes make some of the same venoms that are found in rattlesnakes, and those venoms are just as potent, molecule for molecule. While garter snakes may not have fangs, they can use their tiny teeth to puncture a frog's delicate skin, through which the venom can enter their prey's body. The venom of garter snakes had gone undiscovered until Fry's investigation, simply because people didn't get hurt by their bite.

Fossils and studies on reptile DNA indicate that snakes evolved about 60 million years ago. Their closest living relatives include iguanas and monitor lizards, such as the Komodo dragon, the biggest lizard alive today. Given the ancient origin of some snake venoms, Fry now wondered whether snake venom evolved before there were snakes. He tracked down lizards closely related to snakes and discovered that many of them had glands on the sides of their jaws. He gathered RNA transcripts from those glands and sequenced them. Some of the transcripts were produced from genes closely related to venom genes in snakes. The same aorta-relaxing venom made by inland taipans, for example, is closely related to a protein produced in the mouth of the Komodo dragon (Fry et al. 2006).

Fry has proposed a hypothesis for the evolution of snake venom (FIGURE 8.7). Venom first evolved more than 200 million years ago in the common ancestor of snakes and their closest living relatives. (More distantly related lizards, such as geckos and skinks, produce no venom.) Genes with other functions were recruited for the first venoms, which the early lizards produced in mucus glands in their mouths. These early venoms were not fatal. Some of them were able to slow down the prey of lizards, and others may have caused their wounds to bleed more. When snakes lost their legs about 60 million years ago, they already had a number of venom genes. Later, some lineages of snakes evolved stronger venoms as well as hollow fangs to improve the delivery of their venom. Rather than simply slowing down their prey, these snakes could now use venom to kill them outright. What looks to us today like a complex adaptation made up of parts that can't work on their own was actually assembled, bit by bit, over many millions of years.



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FIGURE 8.7

Fry and his colleagues discovered that some lizards have genes closely related to snake venom genes. Each pink bar shows when one of these genes evolved. This discovery suggests that the complex venom system in snakes began evolving millions of years before snakes evolved. (Information from Fry et al. 2006.)

If Fry is right, there's an unexpected answer to the question, "Which came first, the venom or the fang?" The answer is the venom. In fact, the venom may have come even before the snake.

New Anatomies for Old Bodies

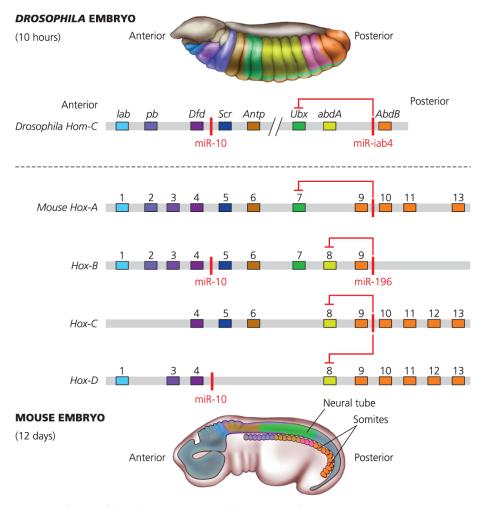
For thousands of years, naturalists have wondered how animals develop into such a vast variety of forms—from legless snakes to five-legged starfish to winged butterflies. In the late twentieth century, scientists first began to decipher the genes that control the development of these body plans. They started out by studying the animals that scientists in previous decades had studied most intensely—mice, chickens, and flies. And the scientists got a shock. The same underlying network of genes governed the development of all these organisms, no matter how different they looked.

At first glance, our body seems very different from a fly's. Like all arthropods, a fly has a segmented exoskeleton surrounding a soft interior. The main nerve of its body runs along the bottom of its abdomen, while the main structures of its digestive system run along the top of its back. It grows segmented legs, along with wings, and a pair of flight-controlling clubs called halteres. It feeds with three pairs of segmented mouthparts, and its eyes are made up of many hexagonal columns, each capturing an image of a tiny fragment of its surroundings.

A vertebrate, such as a human or mouse, has a profoundly different anatomy. Instead of an external skeleton, its skeleton grows inside its body and is surrounded by muscle and skin. Its spinal cord runs down its back, and its digestive system runs down along the abdomen. It grows four limbs; its jaws are internal bones rather than leg-like structures. Its eyes are like little cameras, each able to form a complete detailed image.

The differences between vertebrates and arthropods—along with other major groups of animals—seemed so vast to early naturalists that they doubted these creatures could have evolved from a common ancestor. Their body plans seemed to be separated by a vast gulf. But when scientists uncovered the genes that built those body plans, they realized there were homologies hidden underneath the outward differences. These different animal body plans were built with the same basic "genetic toolkit" (Shubin, Tabin, and Carroll 2009).

When a fly or mammal embryo develops, for example, it differentiates into sections running from the head to the tail. A set of genes called *Hox* genes guide this development in flies. If one of these genes mutates, the head-to-tail anatomy can be dramatically transformed. Legs may sprout out of a fly's head, for example. In the 1980s, scientists discovered that mammals have their own version of *Hox* genes—four sets of them, to be exact. Each set of *Hox* genes is arrayed along a mouse chromosome in the same order in which it is expressed from head to tail in the body. So are the genes in the fly genome. (FIGURE 8.8).



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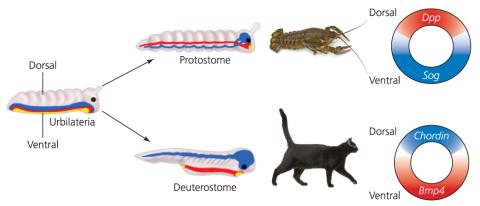
FIGURE 8.8

Flies and mice are separated by more than 570 million years of evolution. And yet the development of each species is controlled by homologous *Hox* genes. In the vertebrate

lineage, the entire *Hox* gene cluster was duplicated twice, producing four sets of genes. Some of these genes were later lost. Yet the overall similarity of *Hox* genes in mice and flies is still clear. Both animals even have homologous genes in the same location within the *Hox* cluster that encodes RNA molecules that regulate other *Hox* genes (marked here as miR). The best explanation for this structural similarity is that the common ancestor of flies and mammals already had a set of *Hox* genes that controlled development. (Information from <u>De Robertis 2008</u>.)

Our understanding of the similarities between the *Hox* genes in flies and in mice has grown greater the longer scientists have studied them. In 2004, for example, David Bartel of MIT and his colleagues discovered micro RNA (miRNA) molecules that map to the *Hox* cluster of genes in mice. These RNA molecules keep some of the other *Hox* genes shut down in certain regions of the developing embryo. Flies, it turns out, have miRNA molecules nearly identical to those of mice, and the genes that encode them are located in the same location in the fly *Hox* cluster. And, like the mouse RNAs, the fly RNAs silence other *Hox* genes. The *Hox* genes are so similar in mice and in flies, in fact, that they are literally interchangeable. If a scientist shuts down a *Hox* gene in a fly and inserts the corresponding gene from a mouse, the fly will develop normally (Bachiller et al. 1994; Lutz et al. 1996).

Flies and mice use similar genes to build their bodies not only from head to tail but also from top to bottom. In a developing mouse, cells along the belly express *Bmp4*. Flies express a homologous gene called *Dpp*, but they express it along the back (**FIGURE 8.9**). The proteins of these genes determine on which side of the body the digestive system will develop. On the opposite side of each embryo, the nervous system will develop (<u>De Robertis 2008</u>).



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FIGURE 8.9

In vertebrates, the gut and spinal cord run along opposite sides of the body. Insects and many other invertebrates have the reverse arrangement. But they use homologous genes to mark where these structures will grow. *Bmp4* is homologous to *Dpp*, and *Chordin* is homologous to *Sog*. These homologous genes are named differently merely because they were discovered independently by different groups of researchers working with different groups of animals. (Information from <u>De Robertis 2008</u>.)

Flies and mice are not the only animals that share this homology in their development. So do octopuses, starfish, oysters, and earthworms. In fact, millions of species of animals use the same system of genes to determine the coordinates of their bodies. They all belong to the same lineage of animals, known as bilaterians. (The name means "two sides," which refers to the symmetry of the left and right sides of their bodies.) The common ancestor of all living bilaterians—sometimes called the urbilaterian—lived some 570 million years ago. It now appears that this common ancestor had already evolved networks of body-patterning genes that laid down coordinates in the embryo.

Turning Fins to Limbs

About 430 million years ago, paleontologists have found, the first vertebrates with paired fins appeared. Today, living fishes sport a variety of fins, from the cartilage appendages of sharks to the bony fins of salmon and trout. But they all use the same network of genes to grow their fins as embryos. We've already met some of those genes before, such as *BMP* and *Hox* genes. These genes, which mapped the body coordinates of our urbilaterian ancestors, were later co-opted to build fins.

In <u>Chapter 4</u>, we explored the fossil evidence for the transition from fins to our own tetrapod limbs. It's also enlightening to look at the genes that build our arms and legs. Many of those genes are identical to the ones that build fish fins. Of course, there's a big difference between your hand and a goldfish fin. Those differences become visible as fish and tetrapod embryos develop. Both start out as little bumps, inside of which cells express *Hox* genes and other genes that lay down a pattern for tissues to follow as they later develop. In fish, a small cluster of bones develop near the base of the fin, most of which develops into a stiff flap of fin rays. In tetrapods, on the other hand, long bones develop, followed by digits. They develop no fin rays at all.

This evidence led to a hypothesis: mutations to the fin gene network expanded the region where bones developed and shrank down the region where fin rays developed. In the 1990s, scientists discovered one especially tantalizing difference in the genes expressed in developing fins and limbs. It involves a gene known as *Hoxd13a*.

In the developing fish fin, the gene produces proteins along the outer rim early in development. The proteins bind to other genes and switch them on, triggering a cascade of activity—some genes are expressed, others repressed—that helps guide the development of the fin. The *Hoxd13a* gene then becomes silent in fish embryos. Likewise, in tetrapods, *Hoxd13a* also becomes active in the early development of limbs and then becomes silent. But after a few days, the gene becomes active once more. It switches back on along the rim of the limb bud a second time. This second wave of *Hoxd13a*

marks a new set of coordinates that will guide the development of digits and wrist bones.

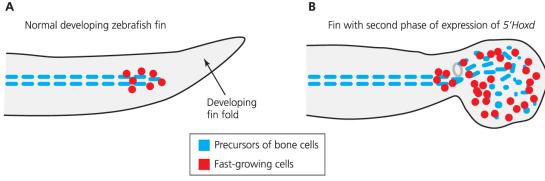
Here, some scientists proposed, might be an important clue to how the hand evolved. It was possible that mutations in our ancestors caused *Hoxd13a* to turn back on again late in development. As a result, it might have added new structures at the end of its fins. If this were true, it would mean that some of the genetic wherewithal to build a primitive hand was already present in our finned ancestors. All that was required was to assign some genes to new times or places during development. Perhaps, some scientists speculated, fishes today might still carry that hidden potential.

Recently Renata Freitas, of Universidad Pablo de Olavide in Spain, and her colleagues set out to try to unlock that potential (<u>Freitas et al. 2012</u>). They engineered zebrafish with an altered version of the *Hoxd13a* gene, which they could switch on whenever they wanted by adding a hormone to a zebrafish embryo.

The scientists waited for the fishes to start developing their normal fin. The fishes expressed *Hoxd13a* early and then became quiet again. A few days later, they added the hormone to the embryos, switching on *Hoxd13a*. This manipulation had a dramatic effect on the zebrafish fin. Its fin rays became stunted, and the end of its fin swelled with cells that would eventually become cartilage.

One of the most interesting results of this experiment is that the extra expression of *Hoxd13a* produced two major effects at once. It simultaneously shrank the outer area of the fin where fin rays develop and expanded the region where bone grew. In the evolution of the hand, these two changes might have occurred at the same time (FIGURE 8.10).





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FIGURE 8.10

A: This illustration shows the structure of a developing fin in a fish embryo. B: When 5' HoxD is expressed late in the development of the fin, its development takes on a striking resemblance to a tetrapod limb. (Information from Freitas et al. 2012.)

It would be wrong to say that Freitas and her colleagues have reproduced the evolution of the hand with this experiment. We did not evolve from zebrafishes. They are our cousins, descending from a common ancestor that lived 400 million years ago. Ever since that split, they've undergone a great deal of evolution, adapting to their own environment. As a result, a late return of *Hoxd13a* in their fins interfered with other proteins in the embryos, and they died.

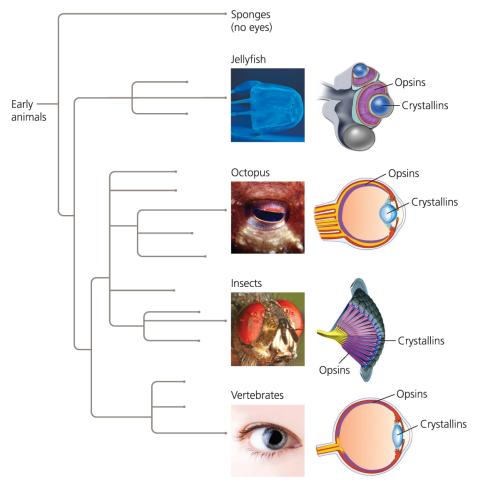
Instead, this experiment provides a clue and a surprise. It provides some strong evidence for one of the mutations that turned fins into tetrapod limbs. And it also offers a surprise: after 400 million years, our zebrafish cousins still carry some of the genetic circuits we use to build our hands.

Evolving Eyes

"The eye to this day gives me a cold shudder," Charles Darwin once wrote to a friend. If his theory of evolution was everything he thought it was, a complex organ such as the human eye could not lie beyond its reach. And no one appreciated the beautiful construction of the eye more than Darwin—from the way the lens was perfectly positioned to focus light onto the retina to the way the iris adjusted the amount of light that could enter the eye. In *The Origin of Species*, he wrote that the idea of natural selection producing the eye "seems, I freely confess, absurd in the highest possible degree."

For Darwin, the key word in that sentence was *seems*. If you look at the different sorts of eyes out in the natural world and consider the ways they could have evolved, Darwin realized, the absurdity disappears. The objection that the human eye couldn't possibly have evolved, he wrote, "can hardly be considered real" (<u>Darwin 1859</u>).

Ever since, the evolution of the eye has intrigued biologists. And in just the past two decades, researchers have begun to tease apart the changes to eyebuilding genes that gave rise to the first eyes hundreds of millions of years ago and produced the staggering diversity of eyes animals use today (FIGURE 8.11).



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FIGURE 8.11

Complex eyes have evolved in several different lineages of animals. Each kind of eye contains crystallins for directing incoming light and opsins for capturing it. But the particular molecules each kind of eye uses as opsins and crystallins are different from those of the others.

All animal eyes capture light with cells known as photoreceptors. But the shapes of these photoreceptors take very different forms in different clades, and they grow inside of eyes with an even greater diversity of shapes. Fly eyes are built out of columns. Scallops have a delicate chain of eyes peeking out from their shells. Flatworms have simple light-sensitive spots. Octopuses and squids have camera eyes like we do, but with some major differences.

The photoreceptors of octopuses and squids point out from the retina, toward the pupil. Our own eyes have the reverse arrangement. Our photoreceptors are pointed back at the wall of the retina, away from the pupil.

To capture light, our photoreceptors use a molecule called opsin. When a photon hits an opsin, it twists, and that change triggers a series of chemical reactions that ultimately lead to an electrical message being sent from the eye to the brain. Biologists have long known that all vertebrates carry the same basic kind of opsin in their eyes, known as a c-opsin (*c* is short for *ciliary*). All c-opsins have the same basic molecular shape, whether they're in the eye of a shark or the eye of a hummingbird. All c-opsins are stored in a stack of disks, each of which grows out of a hairlike extension of the retina called a cilium. In all vertebrates, c-opsins relay their signal from the stack of disks through a pathway of proteins called the phosphodiesterase pathway. All of these homologies suggest that c-opsins were present in the common ancestor of all living vertebrates.

Other bilaterians—such as insects, octopuses, and scallops—don't have copsins in their eyes. Instead, they build another molecule, known as an ropsin (r is short for rhabdomeric). Instead of keeping r-opsins in a stack of disks, they store r-opsins in foldings in the membranes of photoreceptors. All of the r-opsins send their signals through the same pathway of proteins (a different pathway than c-opsins use to send signals in vertebrates). Again, the homologies in r-opsins suggest they evolved in the common ancestor of insects, scallops, octopuses, and other invertebrates that have r-opsins in their eyes.

These dramatic differences led many scientists in earlier generations to conclude that eyes evolved convergently—that many lineages of animals independently evolved organs for capturing images. Yet there is a deep unity to the eyes of the animal kingdom, stretching back some 700 million years.

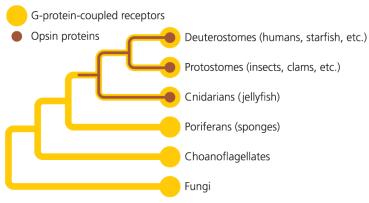
It turns out, for example, that humans make both c-opsins and r-opsins. We don't make r-opsins on the surfaces of photoreceptors where they can catch light. Instead, our r-opsins help to process images captured by the retina before they're transmitted to the brain.

It also turns out that invertebrates have c-opsins. In 2004, Detlev Arendt of the European Molecular Biology Laboratory and his colleagues found them in an animal known as a ragworm. (Arendt et al. 2004). The c-opsins were

not in its eyes, however. They are produced in a pair of organs atop the ragworm's brain.

Findings like these have led researchers to revise their hypothesis about the origin of opsins: both r-opsins and c-opsins were present in the common ancestor of all bilaterians. This hypothesis naturally raised a new question: did the evolution of opsin start earlier than that? To determine the origin of opsins, Davide Pisani of the National University of Ireland and his colleagues conducted a large-scale search for opsin genes in the animal kingdom, as well as for related genes in the relatives of animals (Feuda et al. 2012).

The ancestors of opsins were known as G-protein-coupled receptors (GPCRs). GPCRs are produced by a wide range of eukaryotes, including animals, plants, fungi, and protozoans. They act as sensors, grabbing certain molecules passing by and then relaying a signal inside their cell. Yeast cells, for example, use them to detect odor-like molecules called pheromones that are released by other yeast cells (<u>Jékely 2003</u>; <u>FIGURE 8.12</u>).



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FIGURE 8.12

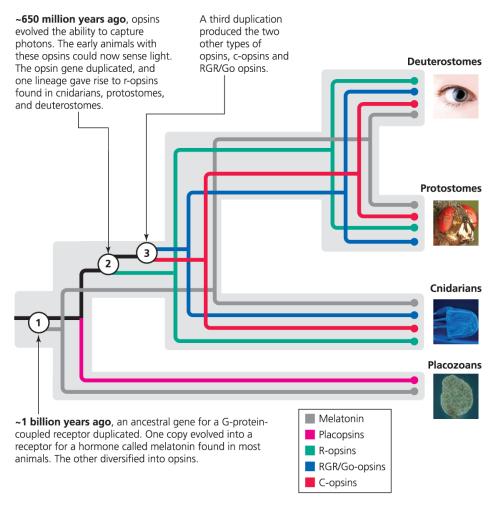
The opsins in animal eyes evolved in the common ancestor of jellyfish, insects, and humans. They evolved from a family of proteins known as G-protein-coupled receptors, which are carried by all animals as well as related organisms such as fungi. (Information from Oakley and Pankey 2008.)

New GPCRs evolved through gene duplication. A mutation produced an extra copy of a GPCR gene, which could then undergo further mutations,

altering its sensitivity to signals. The most closely related GPCR, according to Pisani's research, is a receptor for a hormone called melatonin, which regulates our daily cycle of sleep and wakefulness. Early in the evolution of animals, the ancestor of opsins and melatonin receptors duplicated, producing the two different types of sensors.

To determine where in animal evolution this split took place, Pisani and his colleagues looked for opsins in every major branch of animals. They failed to find opsins in our most distant animal relatives, the sponges. But they did find them in enigmatic animals known as placozoans, which are little more than tiny sheets of cells that hug the seafloor. No one knows what signal placozoan opsins sense. But one thing is clear: they don't have the necessary structure to catch light.

After placozoans split off from our own ancestors, opsins duplicated two more times. The first split gave rise to the r-opsins and c-opsins, which can be found not just in bilaterians, but in cnidarians—a group that includes jellyfish, corals, and anemones. In both lineages, these opsins took on the duty of catching light. But in some lineages, one type or the other evolved to take on other functions, such as the r-opsins in our own eyes (FIGURE 8.13).



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Top to bottom: Monika3steps/Shutterstock; Stana/Shutterstock; Daleen Loest/Shutterstock; Eitel M, Osigus H-J, DeSalle R, Schierwater B (2013) Global Diversity of the Placozoa. PLoS ONE 8(4): e57131.

FIGURE 8.13

Recent studies on opsins show that they evolved from a single common ancestor perhaps a billion years ago. Gene duplication gave rise to different types of opsins that have taken on different functions in the eyes and nervous systems of animals. (Information from Feuda et al. 2012.)

The earliest animals probably produced these opsins in simple light-sensitive eyespots. Such eyespots are found today on many animals, which use them only to sense changes from light to dark. Only later did some animals evolve eyes that can perceive images. In many species, these image-forming eyes also contain transparent proteins called crystallins that can

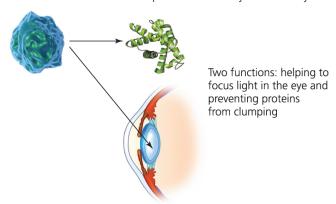
focus incoming light. We have crystallins in the lenses of our eyes, for example. Insects also have crystallins, which fill the columns that make up their compound eyes. To distinguish one group of crystallins from another, scientists identify them with Greek letters— α -crystallins, β -crystallins, and so on.

In the 1990s, scientists began deciphering the evolution of crystallins. It turns out that crystallins also evolved from recruited genes. Consider α -crystallins, a type of crystallin found in all vertebrate lenses. They evolved from proteins that served an entirely different function: delivering first aid for cells (FIGURE 8.14). When cells get hot, their proteins lose their shape. They use so-called heat-shock proteins to cradle overheated proteins so that they can still carry out their jobs. Scientists have found that α -crystallins started out as heatshock proteins. In fact, they still can be found in cells outside the eyes, acting as heat-shock proteins.

1. Small heat-shock protein is expressed in muscles and other tissues.



2. A mutation also causes it to be expressed in the early vertebrate eye.



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FIGURE 8.14

Crystallins (blue) in the lens of the human eye evolved through gene recruitment. At first they carried out other functions in the body, such as preventing proteins from clumping. Mutations caused these proteins to be produced in the eye as well, where they helped to focus images.

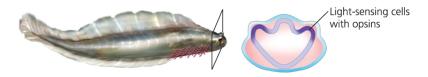
This evidence indicates that in an early vertebrate, a mutation caused α -crystallins to be produced on the surface of their eyes. It turned out to have the right optical properties for bending light. Later mutations fine-tuned α -crystallins, making them better at their new job (<u>Piatigorsky 2007</u>).

Trevor Lamb of Australian National University and his colleagues have synthesized these studies and many others to produce a detailed hypothesis about the evolution of the vertebrate eye (FIGURE 8.15). The forerunners of vertebrates produced light-sensitive eyespots on their brains that were packed with photo-receptors carrying c-opsins. These light-sensitive regions ballooned out to either side of the head and later evolved an inward folding to form a cup. Early vertebrates could then do more than merely detect light: they could get clues about where the light was coming from. The ancestors of hagfish branched off at this stage of vertebrate eye evolution, and today their eyes offer some clues to what the eyes of our own early ancestors would have looked like (Lamb, Pugh, and Collin 2008).

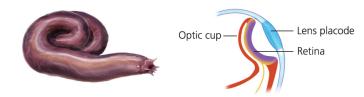
1. Early chordates had light-sensitive eyespots expressing photoreceptor genes.



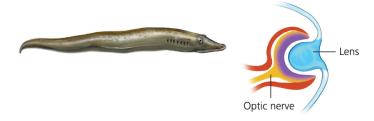
2. Light-sensitive regions bulge outward to the sides of the head.



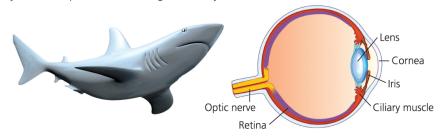
3. Patch folds inward into a cup, beneath unpigmented skin (lens placode).



4. Surface becomes transparent, and lens evolves ability to focus an image.



5. Eyes become spherical and evolve greater acuity.



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FIGURE 8.15

This figure illustrates one recent hypothesis for how the vertebrate eye evolved. It started out as a simple light sensor and then gradually evolved into precise, image-forming

organs. Living vertebrates and relatives of vertebrates offer clues to how this transformation took place. (Information from <u>Lamb et al. 2008</u>.)

After hagfish diverged from the other vertebrates, Lamb and his colleagues argue, a thin patch of tissue evolved on the surface of the eye. Light could pass through the patch, and crystallins were recruited into it, leading to the evolution of a lens. At first the lens probably focused light quite crudely. But even a crude image was better than none. A predator could follow the fuzzy outline of its prey, and its prey could flee at the fuzzy sight of its attackers. Mutations that improved the focusing power of the lens were favored by natural selection, leading to the evolution of a spherical eye that could produce a crisp image.

The evolution of the vertebrate eye did not stop there. Double lenses evolved in some fish, for example, allowing them to see above and below the water's surface at the same time. The ability to see in ultraviolet light evolved in birds. But the huge diversity of vertebrate eyes was nothing more than variations on the basic theme established half a billion years ago.

Constraining Evolution

In this chapter, we've seen how gene duplication and gene rewiring can help produce a staggering range of adaptations. And yet nature is also notable for the adaptations it is missing. Why aren't there any hawk-sized dragonflies? Why are there no nine-toed tetrapods? One explanation for the absence of such forms is that life evolves within constraints. There may be some directions in which evolution rarely—if ever—travels (Maynard Smith et al. 1985).

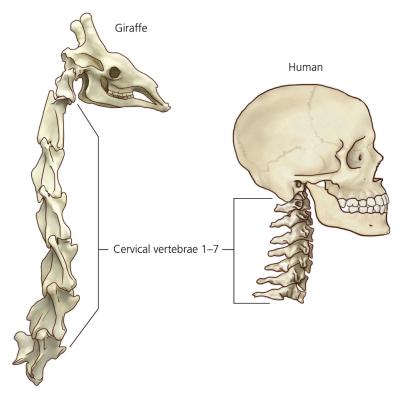
The laws of physics can impose constraints that evolution cannot overcome. Animals such as elephants and sharks can reach enormous sizes, for example, and yet the most diverse clade of animals, the insects, is relatively small. The reason there are no gigantic insects alive today, a number of researchers have argued, is that the concentration of oxygen in today's atmosphere makes it impossible. Insects get oxygen through tiny tubes penetrating their exoskeletons. As the insects become bigger, they need more tubes to supply enough oxygen to support their larger bodies. But as an insect gets bigger, the tubes become a less efficient way to deliver oxygen to its tissues. As a result, the tubes themselves must get bigger in order to deliver sufficient oxygen to power the insect's muscles, and there's less room for muscles and other structures that support the animal.

It's possible to test this hypothesis by looking back through the history of life to times when the oxygen concentrations were higher. If insects can absorb oxygen more efficiently, they don't require larger tubes, and they can evolve to larger body sizes. It just so happens that during the Carboniferous period, oxygen concentrations were much higher than they are today (35 percent compared to the current 21 percent). Paleontologists have found remarkably large fossils of flying insects from that period—one dragonfly-like species called *Meganeura*, for example, reached the size of a seagull (Harrison, Kaiser, and VandenBrooks 2010; Kaiser et al. 2007).

Populations may also be constrained from evolving in certain directions by pleiotropy. A mutation may have a beneficial effect on one trait but have a harmful one on another. In <u>Chapter 6</u>, we saw how mosquitoes can rapidly

evolve resistance to pesticides, but resistance mutations also make them more vulnerable to predators. Pleiotropy is particularly good at constraining evolution in the development of animals. A single gene, such as a *Hox* gene, may help guide the development of many different structures in an embryo.

The harmful effects of mutations on developmental genes may explain some of the limits of adaptations found in complex species. Almost all mammals, for example, have exactly seven cervical vertebrae in their necks: the only exceptions are sloths and manatees. There's no obvious reason that natural selection should favor seven neck vertebrae over six or eight. Even giraffes have only seven cervical vertebrae, despite having the longest necks of all mammals (FIGURE 8.16).



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FIGURE 8.16

Almost all mammals have only seven cervical vertebrae in their neck. This rule applies even to species with long necks, such as giraffes. This pattern may be the result of evolutionary constraint. Mutations that would lead to more or fewer cervical vertebrae may have deleterious effects as well.

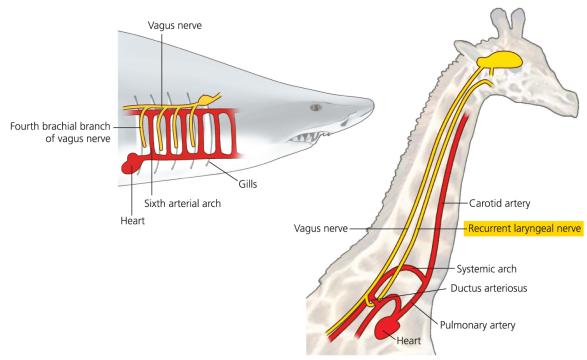
Frieston Galis of Leiden University and her colleagues wondered if some type of strong pleiotropy was responsible. Perhaps a mutation that might add or subtract vertebrae would have some harmful side effect. On rare occasion, children are born with an abnormal number of cervical vertebrae, and so Galis and her colleagues searched through Dutch medical records to see if these cases were accompanied by any other disorders. They found that fetuses with abnormal necks were more likely to be stillborn; if the children survived past birth, they were 120 times more likely to develop pediatric cancers (Galis et al. 2006; Varela-Lasheras et al. 2011).

Galis and her colleagues have also found fitness costs for abnormal numbers of cervical vertebrae in mice and other animals. Two mammal species that break the rule—sloths with eight cervical vertebrae and manatees with six—have very low metabolisms for a mammal. Low metabolic rates have been linked to low rates of cancer. It's thus possible that fitness cost of evolving more or less cervical vertebrae disappear in slow-moving mammals.

Constraints do more than block certain paths of evolution. They also build quirks and weaknesses into new adaptations. New adaptations do not appear out of the blue. They're modifications of what's already there. Fish, for example, grow a series of nerve branches from their spinal cord that extend into their gill pouches. Tetrapods evolved from lobe-fins, inheriting this basic arrangement of nerves. But as the tetrapod neck evolved, the ancestral gill arches shifted their positions and their sizes. The nerves migrated with the arches, stretching into peculiar paths.

The most spectacular of these detoured nerves, the recurrent laryngeal nerve, extends down the neck to the chest, loops around a lung ligament, and then runs back up the neck to the larynx (FIGURE 8.17). In long-necked giraffes, the nerve grows to a length of 20 feet in order to make this U-turn, when 1 foot of nerve would have done quite nicely. But a 1-foot nerve never arose in giraffes, because this type of variation never existed. Ancestral giraffe populations contained individuals with nerves that developed according to their ancient, inherited developmental mechanism, and this mechanism was incapable of producing a simple, 1-foot nervous connection to the larynx. History dictated the possible variations of the giraffe's

recurrent laryngeal nerve, and selection could operate only on the variation that history provided.



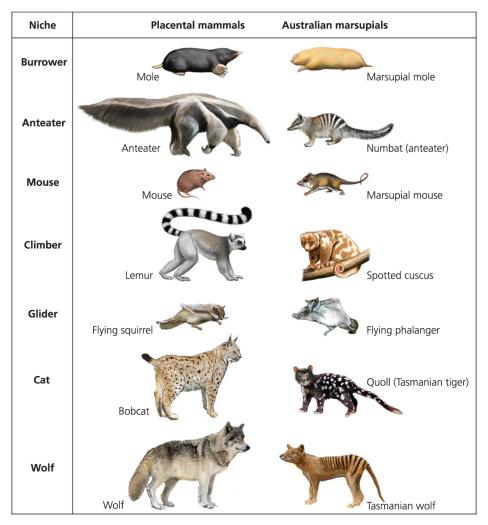
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FIGURE 8.17

All tetrapods evolved from aquatic vertebrate ancestors. Those ancestors had nerves that extended back from the brain to the gill arches, crossing over blood vessels. As tetrapods adapted to life on land, they lost functional gills but retained the ancestral organization of gill pouches in their neck. Their nerves continued to loop around blood vessels, as they had in fishes. An extreme case of this evolutionary constraint can be found in giraffes. The recurrent laryngeal nerve travels all the way down the giraffe's neck, where it loops around a blood vessel and then returns all the way back up, to a spot just a few inches from its origin. (Information from Dawkins 2009.)

Convergent Evolution

The cougars and wolves that roam North America are placental mammals. In other words, female cougars and wolves carry their developing fetuses in their uterus, where an organ called the placenta helps the fetuses grow. Cougar-like and wolf-like animals also once roamed Australia, but they were of a different sort. They were marsupial mammals, which do not grow placentas. Instead, their young crawl out of the uterus and into a pouch to finish developing. Marsupial and placental mammals diverged from a common ancestor about 130 million years ago, and both lineages eventually produced species that looked remarkably similar and occupied the same ecological niches (FIGURE 8.18).



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FIGURE 8.18

Marsupials and placental mammals diverged from a common ancestor 130 million years ago. But they have repeatedly evolved body plans that show remarkable convergence. This convergence is the result of adaptation to the same ecological niches, such as burrowing underground or attacking large prey.

The evolution of two lineages into a similar form is known as convergent evolution. Two distantly related lineages will often evolve into the same form by different routes. Dolphins, sharks, and tuna have similar bodies, allowing them to move efficiently in water. Unlike sharks and tuna that retain basic fin structures from their aquatic relatives, fishlike bodies evolved in dolphins with the loss of their hind legs and many other modifications to their

terrestrial mammal body. Even though all three are excellent swimmers, dolphins retain some clues to their terrestrial ancestry. While sharks and tuna swim by undulating from side to side, dolphins, like their mammal relatives that gallop by flexing the spine up and down, swim by moving their tails up and down.

The developmental programs that build dolphins and sharks are profoundly different, despite the similar phenotypes they generate. To pick just one of many examples, dolphins, as embryos, begin to develop hindlimb buds that then stop growing as the rest of their body gets larger (page 14); sharks lack the genetic circuits necessary to produce tetrapod limbs in the first place.

In other cases of convergence, lineages have evolved identical phenotypes by independently acquiring mutations on the same genes. This kind of evolution is known as parallelism. One of the best-documented cases of parallelism involves stickleback fish. Populations of sticklebacks that live in the ocean typically produce armor plates and sharp spines to ward off predators. Toward the end of the last ice age, about 15,000 years ago, some populations of sticklebacks moved up rivers and into lakes. There they enjoyed a predator-free life. In each lake, the sticklebacks independently lost much of their armor. Some have lost their spines entirely.

David Kingsley, a biologist at Stanford University, and his colleagues have uncovered the genetic changes that produced this parallelism. They gathered sticklebacks from lakes in California, Washington, British Columbia, and the Northwest Territories of Canada, as well as in Iceland and Scotland. They bred these lake sticklebacks with their heavily armored and spiked cousins from the ocean. Some of their offspring developed armor and spikes; some didn't. By comparing the DNA of these hybrids, Kingsley and his colleagues discovered that, in every lake they studied, mutations to the same gene (called *Eda*) had led to the reduction of armor (Shapiro et al. 2004).

The researchers also discovered that all the sticklebacks that grew spines inherited the same segment of DNA from their marine parent. Fish lacking spines had inherited the same segment from the freshwater parent. When the team examined that segment closely, they discovered that a gene called *Pitx1* was essential for the development of the spines. Yet the sequence of *Pitx1* was identical in both the marine and freshwater sticklebacks.

The only difference between the two kinds of fish is where *Pitx1* is expressed. In freshwater sticklebacks, *Pitx1* is expressed in the developing nose, thymus gland, and sensory neurons. Marine sticklebacks express the same gene in all those cells, as well as in cells that will develop into spines. It thus appears that in freshwater sticklebacks, a mutation has disabled one binding site near *Pitx1* that makes it become active in the spines. The other binding sites—along with the gene itself—are unchanged.

As scientists probe deeper into the developmental programs that have evolved in animals, it gets harder to draw a clean line between convergence and parallelism. Eyes, once again, provide an excellent example. Complex eyes can be found in many animals. In each case, they contain lenses to focus light on photoreceptors. But that does not mean that these animals share a common ancestor that also had a complex eye. The octopus eye, for example, is radically different from our own. For one thing, its receptors face forward, not backward. The crystallins that make up our lenses were borrowed from different genes than those in octopuses. So, on one level, the complex eye is a case of convergent evolution.

But all complex eyes also share a deep ancestry. Their opsins evolved from the same ancestral opsin. The development of all complex eyes in bilaterians is controlled in part by the same gene, known as *Pax-6*. When scientists inserted the *Pax-6* gene from a mouse into a fly's genome, the fly sprouted extra eyes over its body.

Is the fly eye homologous to the mouse eye, or is it convergent? The answer is both, depending on which level you look at.

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Sexual selection has produced many of the most striking traits in animals, such as the antiers of this bull elk.

Being a scientist can mean learning to do some pretty strange things. For Patricia Brennan, an evolutionary biologist at the University of Massachusetts, those strange things include measuring the length of a duck's phallus—the duck equivalent of a human penis. The task is made all the more difficult because a male duck keeps his phallus retracted inside his body, extending it only during mating. To measure it is therefore a two-person job.



Bernard J. Brennan.

FIGURE 9.1

Patricia Brennan has discovered that an evolutionary arms race between male and female birds can produce gigantic sexual organs.

Brennan has a colleague grab a bird and hold it upside down, its legs sticking out in the air. If the maneuver is done with care, the duck does not quack or struggle; it just gazes off into the distance. Brennan gently presses around a small dome of muscle below the bird's tail, and after a little coaxing the phallus emerges. Brennan grabs a ruler.

The measurement Brennan makes depends on the time of year. After the breeding season is over, a duck's phallus shrinks down to a tiny length. When the next breeding season approaches, it grows to astonishing lengths. In some species of ducks, it can measure as long as the bird's entire body.

The length of a duck's phallus is all the more remarkable when you consider that only 3 percent of all bird species have phalluses at all. (In the other species, the male has only a simple opening that he positions against a similar opening in the female.) Brennan wants to understand why duck phalluses are so elaborate, especially when other bird species have none.

To discover why, she has embarked on a study of the forces driving their evolution.

A male duck's phallus is a spiral-shaped organ that twists counterclockwise. But Brennan found that in ducks and other waterfowl, the female's reproductive tract—called an oviduct—twists clockwise. To see the effect of these mismatched twists, Brennan has figured out how to watch duck phalluses in action. She designed twisting glass tubes of the same size and shape as duck oviducts. She took her bizarre sculptures to a California duck farm, where the workers are skilled at collecting the sperm from prize male ducks to use for breeding. There, she coaxed the male ducks to mate with the glass tubes.

First she had them insert their phalluses into glass tubes that had the same counterclockwise twist. Brennan found that the duck phalluses expanded to the end of the tube in just a third of a second. Next, she tried out glass tubes that twisted clockwise, the way real duck oviducts do. Now the male ducks could push their phalluses slightly into the tube. Brennan's experiment shows, strangely enough, that the reproductive tract in female ducks actually hampers mating, rather than helping the process along (Brennan, Clark, and Prum 2010).

Brennan's research is part of a growing body of work, carried out on many species, that points to a surprising conclusion: adaptations often evolve in males and females that put one sex in conflict with the other. In the case of ducks, the female reproductive tract thwarts the efforts of males to fertilize the female's eggs.

The reason for this conflict appears to lie in the mating system of ducks. More than one male duck may mate with a female, and so she will end up with the sperm of several males. Thanks to the mismatched twists of the female's oviduct, males can insert their sperm, but only at its opening. In other species, scientists found that females can store sperm from several males in different pouches and then later control which male's sperm will fertilize their eggs. Brennan discovered that the duck's oviducts have rows of pouches along their length. Could ducks be doing the same thing?

The results from another study Brennan carried out suggest that this is indeed the case. She traveled to Alaska, where she caught 16 species of ducks and other waterfowl that migrate there for the summer. After measuring the phalluses on the male birds, Brennan then turned her attention to the females. She found a striking correspondence. In species where males have longer phalluses, the females have oviducts with more pouches and more coils. Her research suggests that the conflict between males and females is more intense in some species than others, and that intensity drives the evolution of extreme genitalia in both sexes.

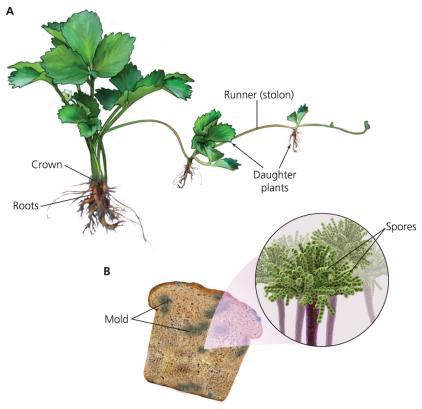
In previous chapters, we've seen how natural selection acts on populations, spreading beneficial alleles and even giving rise to complex adaptations. But Brennan's ducks are a stunning demonstration that life is much more than just surviving the elements and finding food. To pass their genes down to future generations, sexually reproducing organisms must find mates and produce healthy offspring. Sex has produced some of nature's most extravagant traits, from the genitalia of ducks to elk antlers to insect dances.

In this chapter, we'll take a look at sexual selection. We'll also look at the consequences of sex: the offspring that result. Animals have evolved a staggering diversity of strategies for rearing their offspring—from male seahorses that become pregnant to mother birds that abandon their nests. The explanation for this diversity of parenting is much the same as for the diversity of sex: evolution.

Why Sex?

A discussion of sex must start with the very existence of sex itself. Many species do without sex altogether. Bacteria can breed without sexual reproduction, meiosis, or the fertilization of eggs. They simply divide in two. While sex is common among animals, scientists have found some species that also do without sex. Some species of whiptail lizards in the southwestern United States are made up entirely of females, for example. Instead of getting sperm from males, they duplicate the chromosomes in their developing eggs. The twin chromosomes undergo recombination during meiosis, and the egg begins to develop into a lizard embryo (Lutes et al. 2010).

For still other species, sex is optional. Many plants have two options. A strawberry plant, for example, can fertilize its ovules (the plant version of eggs) with pollen (the plant version of sperm). Or it can send out runners, which produce new plants that are genetically identical to it (FIGURE 9.2). Many species of flatworms are hermaphrodites: they develop both male and female organs. They can receive sperm from other flatworms or inseminate their partners. But they also can fertilize their own eggs with their own sperm. Many flowering plants are hermaphrodites, too. A single plant will grow ovules and pollen grains.



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FIGURE 9.2

Many species can reproduce without sex. A: Strawberry plants can grow runners, which produce new plants complete with roots, stems, and flowers. B: Many kinds of fungi produce spores, which produce genetically identical individuals.

All these forms of reproduction are made possible by genes, and those genes are subject to natural selection. And therein lies a puzzle. As an evolutionary strategy, sexual reproduction seems at first glance like a recipe for failure. It imposes huge costs on organisms—costs that could well drive a population extinct.

To appreciate the toll sexual organisms pay, imagine an island full of lizards. Some of the lizards don't need to mate, and some do. Each asexual female can produce many daughters, each of which can produce daughters of her own. Meanwhile, the sexually reproducing females can reproduce only by mating with a male. About half of their offspring will be males, which cannot produce young themselves. Sex, in other words, effectively cuts the

reproducing population of these lizards in half. For the same amount of energy invested, asexual females produce twice as many copies of their genotype as sexual females (FIGURE 9.3). The late British evolutionary biologist John Maynard Smith dubbed the disadvantage the twofold cost of sex (Maynard Smith 1978).

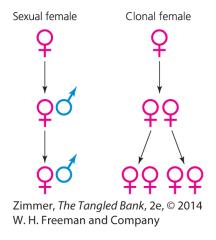


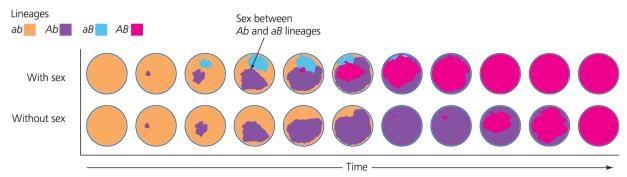
FIGURE 9.3

Generation after generation, an asexual lineage can multiply faster than a sexual one. That's because all the offspring of asexual organisms can reproduce themselves, whereas sexual organisms produce males and females. Sex must have some other evolutionary benefit to explain its widespread existence.

If this cost isn't countered by some benefit, selection would favor genes for asexual reproduction until sex disappears from the island of lizards altogether. Despite this cost, sex is everywhere. Its commonness suggests that sex has some evolutionary benefit that outweighs its cost.

Scientists are exploring several hypotheses about the main benefits of sex. According to one of them, sex speeds up the pace of adaptation. If an asexual organism picks up a beneficial mutation, it can pass the mutation only to its direct offspring (FIGURE 9.4). If two asexual organisms each acquire a different beneficial mutation, the mutations will stay isolated in their respective lineages. Sexual reproduction, on the other hand, combines genotypes in each generation into new combinations (see Chapter 5). Two beneficial mutations can combine in one individual, raising its fitness even more than either could on its own. The harmful mutations, meanwhile, can be

eliminated faster from the population as a whole. In asexual species, there's no way to separate the bad mutations from the good. The bad mutations continue to accumulate, dragging down the average fitness of the population.

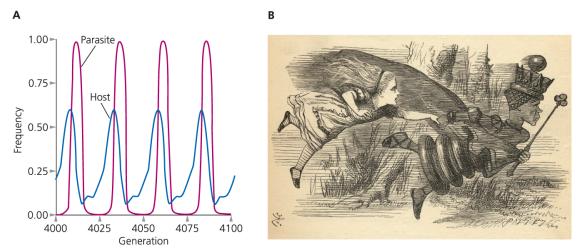


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FIGURE 9.4

One hypothesis for why sex persists is based on its ability to produce new genotypes. These two figures show a sexual (*top*) and asexual population (*bottom*). Each population starts out with a single allele, *a*, for one gene, and a single allele, *b*, for another gene. Over time, a mutation changes *a* to a new allele, *A*, in a single individual. This mutation is beneficial, and so its descendants become more common (purple). Meanwhile, another individual acquires a beneficial mutation to *b*, producing the allele *B* (blue). In a sexual population, *aB* and *Ab* individuals can mate and produce offspring with the *AB* genotype (red), which has an even higher fitness. In an asexual population, on the other hand, the same two mutations must evolve in a single lineage. As a result, it takes longer for the *AB* genotype to evolve.

According to another hypothesis, parasites are responsible for sex (FIGURE 9.5). A parasite can lower the fitness of its host in many ways—by killing its host outright, making it too sick to find a mate, or damaging its reproductive organs. Hosts vary in their defenses to parasites; some have genes that make them especially vulnerable, while others can resist most infections. An allele that helps hosts fight off parasites may raise the hosts' fitness and thus be favored by natural selection.



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FIGURE 9.5

A: According to one hypothesis, parasites drive hosts through a cycle of booms and busts. This graph shows results from a computer simulation for host-parasite coevolution. The blue line is the frequency of one host genotype; the purple line gives the frequency of the parasite genotype that can infect it. Both genotypes oscillate over time, as if they were "running" in circles. Some studies suggest that sex helps hosts evolve fast enough to maintain their defenses against parasites. B: This explanation for sex is called the Red Queen hypothesis, named after the character in Lewis Carroll's *Through the Looking-Glass* who runs very fast just to stay in place.

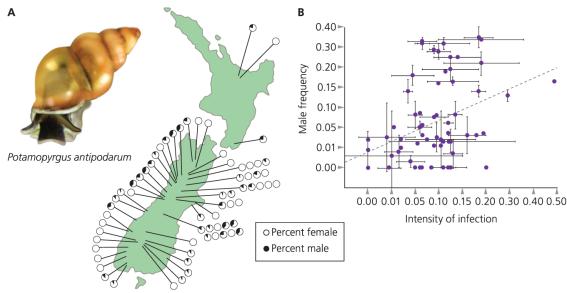
But the parasites are also evolving, and now a mutation that allows them to overcome a host's new defenses will be favored as well. In the 1970s, several evolutionary biologists suggested that parasites and hosts evolve in cycles. First, the parasites would adapt to the most common genotype in the host population. Those hosts eventually would be decimated, and another host genotype would become more common. Now the most common genotype in the parasite population would be poorly adapted to the most common host genotype. Over time, a new parasite genotype would evolve that was better adapted to the new host.

This model of evolution is known as the Red Queen effect. The name comes from the Red Queen in Lewis Carroll's book *Through the Looking-Glass*, who takes Alice on a run that never seems to take them anywhere.

"Now here, you see, it takes all the running you can do to keep in the same place," the Red Queen explained. This model predicts a race between hosts and their parasites, forcing hosts to evolve defenses at a rapid rate just to "stay in the same place"—that is, to survive.

Sex may give organisms an advantage in this race. By mixing together alleles into new combinations, it may generate new genotypes that can deliver resistance to parasites faster than by asexual reproduction.

Mathematical models of the Red Queen effect support this hypothesis, as do a number of studies on real organisms. Curtis Lively, a biologist at Indiana University, and his colleagues have found evidence for the Red Queen effect in a species of freshwater snail in New Zealand called *Potamopyrgus antipodarum* (FIGURE 9.6). Within the species, some snails reproduce asexually and others need to find a mate. In some lakes, all the snails are asexual; in others, all snails are sexual. A main parasite of the snails is a flatworm known as a trematode. In some lakes the trematode is rare, and it's common in others. Lively and his colleagues have found that in lakes where snails face a greater risk of infection by the trematode, sex is more common—exactly as the Red Queen hypothesis would predict (Lively 2010).



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FIGURE 9.6

A: The New Zealand freshwater snail, *Potamopyrgus antipodarum*, can reproduce either sexually or asexually. Curt Lively of Indiana University studies the snails to see which

conditions favor the evolution of each type of reproduction. He surveyed 66 snail populations, tallying the asexual and sexual forms. His results are shown here as the proportion of snails that are males, ranging from 0 percent (all asexual) to 50 percent (all sexual). B: Lively also measured how much the snails were infected with parasitic flatworms called trematodes. He found that where infections are more intense, the proportion of males is higher. This correlation is what you'd expect if parasites favor the evolution of sex. (Data from <u>Lively 1992</u>.)

Cheap Sperm and Costly Eggs

Human eggs are 4000 times larger than sperm. That's a general rule for sex cells (known also as gametes). Not only are male gametes much smaller than female ones, but they're much more abundant. Over a woman's entire lifetime, only about 400 eggs will reach maturity and become ready to be fertilized. Men, by contrast, make tens of millions of sperm every day (FIGURE 9.7).



James Steidl/Shutterstock.

FIGURE 9.7

A sperm fertilizes an egg. Male gametes are small and mobile, while female gametes are large and move relatively little.

Thanks to these differences, the best strategies for boosting reproductive success are different for the sexes. Female success isn't limited by a small supply of sperm. A single man can make enough sperm to fertilize every egg in every woman on Earth. The limit on female reproductive success lies instead within her: the limited number of eggs she can nurture and rear to maturity. Females that do a better job at rearing their young are favored by selection over other females (FIGURE 9.8).



In Green/Shutterstock (left), ClassicStock.com/ClassicStock.com (center), www.raywilsonbirdphotography.co.uk (right).

FIGURE 9.8

In many species, females keep their young inside their body, providing nourishment and protection. Examples include humans (A), zebra (B), and tsetse flies (C).

Since sperm are so easy to make, they don't limit a male's reproductive success. Instead, he faces a scarcity of eggs. Thus, in many species the male's best strategy is to fertilize as many eggs as possible—even if he must mate with many females to do so.

This imbalance explains why males in most animal species compete with each other for the opportunity to mate with females. Their struggles have evolved into a staggering diversity of forms, from mountain sheep slamming their horns against each other to male fiddler crabs flipping each other over with their outsized claws. As males compete for females, some end up having more offspring than others.

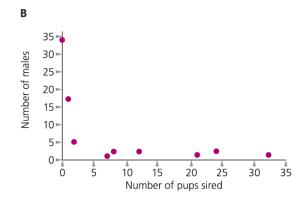
The difference between the winners and the losers is especially stark among the southern elephant seals that breed on Sea Lion Island, one of the Falkland Islands in the South Atlantic Ocean. Male elephant seals fight to mate with large numbers of females, which gather together in groups known as harems. They rear up on their flippers and throw their tremendous bodies against each other, sometimes drawing blood with their teeth. The losers slink away to lurk at the edges of the colony.

A. Rus Hoelzel, a biologist at Durham University in England, and his colleagues surveyed all the seals on the island, year in and year out (<u>Fabiani et al. 2004</u>). They snipped small pieces of skin from all the adult elephant seals as well as from all 192 baby seals that were born in 1996 and 1997.

They could identify the father of almost every seal pup, because the females were mating only with males that also were lying around on Sea Lion Island. Of all the males, 72 percent failed to have any offspring at all. The remaining 28 percent did not have an equal share of reproductive success. Many had only one or two pups, while a few managed to have many offspring. One particularly successful male seal fathered 32 pups.

Hoelzel found that 90 percent of the seals that fathered pups were the heads of harems. The other 10 percent of the seals that fathered pups were lurkers that managed to sneak off with a female. The bigger a male elephant seal, the better his chances of defending a harem and fathering a lot of pups. In other words, large size is selected in the male seals, in much the same way large beaks are selected in medium ground finches on the Galápagos Islands when there are a lot of hard seeds to eat (FIGURE 9.9).





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FIGURE 9.9

A: Male elephant seals fight each other in order to mate with large numbers of females. B: In a typical breeding season, a small number of dominant males achieve disproportionate mating success while most of the males sire no pups at all. (Data from Fabiani et al. 2004.).

The selection for size that takes place among elephant seals is not based purely on how long the seals survive. A trait that boosts the opportunities the seals have to mate is being selected. This special type of selection is known as sexual selection.

Sexual selection may account for why male elephant seals are several times larger than female ones. Bigger males tend to win fights with smaller ones, and so bigger males tend to hold onto harems. The big males thus have more offspring. Female elephant seals, on the other hand, don't fight with each other, and so large females don't have a reproductive advantage over small ones. In other words, sexual selection for body size is much stronger in male elephant seals than in females.

Songs and Dances

The competition of males to mate with females gives the females the opportunity to choose which male they will mate with. In many species, females have evolved strong preferences about mating (FIGURE 9.10). Among birds, for instance, females will often prefer males with complex songs over males with simpler ones. Female frogs will choose males that croak loudly over quiet ones. Females judge males not only on what they do but also on how they look. Male jungle fowl with big combs on their heads have more reproductive success than males with smaller combs. Male swordtail fish develop a long extension on their lower tail fin, and the males with longer swords are more likely to attract females than short-sworded males. One of the strangest effects of female choice can be found in stalk-eyed flies. There are several hundred species of these flies, and in most of them the males have eyes on sideways-pointing stalks that can be longer than their entire body. The distance between their eyes has a strong effect on the preference of the female flies: the longer the stalks, the more likely they are to mate.



ZUMA Press, Inc./Alamy (left); Jill Y Nightingale/Getty Images (center); Rike_/Getty Images (right).

FIGURE 9.10

Male displays can take many forms, from bright plumage to neck flaps to loud, croaking choruses. In many species, females use these displays to choose their mates.

These mating preferences may evolve from ancient preferences that animals have for certain shapes or colors. On the Caribbean island of Trinidad, for example, the male guppies sport bright orange patches on their bodies, and the females prefer to mate with the males with the biggest, brightest patches (FIGURE 9.11). Helen Rodd, a biologist at the University of Toronto, noticed that these patches look a lot like the bright orange fruits that fall into the streams in Trinidad and are eaten by the guppies. She wondered if the males were evolving patterns that mimicked the fruit (Rodd et al. 2002).



Gregory F. Grether.

FIGURE 9.11

Female guppies prefer to mate with males with bright orange spots. Their preference may have originated with an attraction to orange-colored fruits that sometimes land in their streams.

To test the idea, Rodd and her colleagues tossed little orange disks into guppy tanks. They found that the guppies pecked at them more than they did at disks of other colors. Males were as enthusiastic as females, indicating that the female guppies were not confusing orange disks with attractive male fish. What's more, Rodd and her colleagues found that some populations of guppies responded more strongly to orange disks than others, and the

strongest responses came from populations whose females had the strongest preference for orange males.

In 1915, the British biologist Ronald Fisher developed a model to explain how these preferences can drive sexual selection. He started out by envisioning a population in which some females preferred some particular trait in males, and some females were not choosy about their mates. In such a population, the choosy females will mate only with the showy males, and the unchoosy ones are equally likely to mate with any male. Overall, this arrangement means that showy males will have better odds of reproducing than drab ones. As a result, their genes for showy traits become more common in the population.

Meanwhile, the females help drive the evolution too. Females that prefer showy males will produce showy sons—which also will enjoy better odds of reproducing. Thus a strong preference for showy males raises the reproductive success of the females. Fisher argued that the selection acting on both male traits and female preferences would create a feedback loop, which he called a "runaway" mechanism.

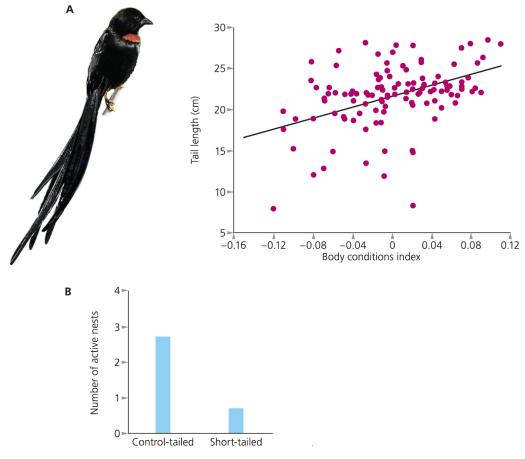
This runaway mechanism could be stopped only when a sexually selected trait started to become a serious threat to a male's survival. Orange patches don't merely attract female guppies, for example; they also attract predatory fish. And studies on guppies suggest that their evolution is limited just as Fisher would have predicted. In streams with predators, the orange patches on male guppies are drab compared with the vibrant guppies that live in predator-free waters.

Another hypothesis holds that sexual selection takes place because displays communicate information from one sex to the other. Animals can use them like billboards to advertise their value. Scientists who favor this explanation point out that the choice of a mate can have a huge effect on an animal's reproductive success. Depending on the quality of a mate's genes, the fitness of an animal's offspring may go up or down. If animals can improve their odds of picking mates with good genes over bad, they can potentially increase their fitness in the long term. But animals can't order genetic tests on their mates. Instead, they can only have a preference for mates with some reliable sign of good genes—be it a song or a spot or a feather.

There is a risk, however, in relying on such signals in choosing a mate. Imagine, for example, that female birds are attracted to blue-headed males because blue-headed males father fitter chicks. Other male birds may gradually evolve blue heads as well, attracting the females even though they don't have high-quality genes. Sexual signals open the door to deception.

One way a sexual display can become a reliable signal is for an animal to have to pay a cost to make it. That cost might be the resources required to grow antlers or the energy it takes to croak all night long. Strong animals can afford that extra energy, but weaker ones cannot. Such costly signals ought to be particularly difficult for weaker animals to make when they're under other kinds of stress, such as sickness or starvation.

These models have inspired some biologists to search for evidence of honest signals in real animals. Sarah Pryke, of the University of New South Wales in Australia, has tested the hypothesis by studying red-collared widowbirds in Africa. Male red-collared widowbirds have tail feathers that measure around 22 centimeters long—longer than their entire body (FIGURE 9.12). During the mating season, they establish territories that they fly around while fanning out their feathers so that they flap in the breeze. Female red-collared widowbirds spend this time choosing where to make their nests, mating with the male whose territory they've picked.



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A: Male red-collared widowbirds have long tails that attract females. As this graph shows, males in the best condition have longer tails than males in poor condition. B: Biologist Sarah Pryke clipped the tails of some males to observe the effect on the attractiveness of the males. She found that artificially short-tailed males ended up with fewer females nesting on their territories.

Males with longer tails, Pryke found, attracted more females to their territories. To see how strong this preference was, Pryke snipped the tails of 120 male red-collared widowbirds. On 60 birds, she snipped their tails down to 20 centimeters (cm); on the other 60, she cut the tails down to 12 cm. Then she compared how well the males fared in the mating game. (Although 12 cm is short for a red-collared widowbird tail, it's still within the natural range of the birds.)

Pryke found some dramatic differences between the two groups of birds. A long tail costs male red-collared widowbirds extra energy because of the drag it creates in the air. Birds with clipped tails were able to spend more time flying around, attracting females. Yet that extra time did not translate into more reproductive success. The birds with the longest tails ended up with three times more nesting females in their territories than the short-tailed birds (Pryke and Andersson 2005).

A long tail, in other words, comes at a cost to male red-collared widowbirds. The cost suggests that it is an honest signal to females. What clues does it give the females? Pryke has found that males with long tails are in better condition than males with shorter tails. By selecting long-tailed mates, female red-collared widowbirds may be picking males that are strong enough to hold their territories and to give their offspring genes for good health.

The evolution of honest signals may account not just for female preferences but also for the horns, tusks, antlers, and other extravagant weapons that male animals can grow. These weapons may be for fights as well as avoiding fights. Battling another male is very risky, potentially leaving both animals wounded or dead. In many species, males reduce this risk by sizing up their opponents from a distance. A big weapon may serve as a clear signal of the strength of the male that carries it, and weaker males may back away at just the sight of it.

Of course, such a signal cannot survive very long if it's easy to fake. If weak males can make big weapons, then natural selection may favor males that ignore the weapons and fight opponents anyway. There's growing evidence, though, that weapons are indeed honest signals.

Douglas Emlen, a biologist at the University of Montana, has been finding evidence for this honesty in male rhinoceros beetles, which grow enormous horns to fight with competitors (FIGURE 9.13). The evidence can be found in the hormones that control the growth of the beetles. In good conditions, the insects produce a lot of hormones, which spur them to grow to large sizes. In bad conditions, low hormone levels leave the beetles stunted. Males with good genes grow to larger sizes than do other males, no matter what the environmental conditions are (Emlen et al. 2012).



Doug Emlen.

Male rhinoceros beetles grow long horns they use to fight with other males. The development of horns is extremely sensitive to growth hormones. Scientists blocked growth hormones in the beetle on the right, causing it to develop a disproportionately small horn. This powerful influence ensures that horns are an honest signal of the quality of males.

Emlen and his colleagues shut off the hormone signals in developing beetles. Then they measured the effect on the insects' bodies, as well as different organs including their horns. Blocking the signal shrank the bodies of the beetles by a modest margin. But the horns were devastated. Compared to the wings of the beetles, Emlen and his colleagues found, horns were eight times more sensitive to the hormone manipulations.

Emlen's study suggests that the exaggerated size of beetle horns is linked to the overall quality of males—and that the link can't be severed. A small beetle can't produce a deceptively big horn, because it needs the same hormone signals it would take to build a big body.

Mating Games

In the animal kingdom, there are many different kinds of mating systems. Mimic poison dart frogs (*Ranitomeya imitator*) will pick mates and then remain absolutely loyal for life. Together, a male and a female will work to raise their young in tiny pools of water that form in bromeliads lining the branches of tropical trees. This mating system is known as monogamy. Among elephant seals, by contrast, a single male has many female mates at a time—a system known as polygyny. Central American birds known as wattled jacanas are at the other extreme, polyandry, in which each female may mate with several males.

Until the late 1900s, naturalists could study mating systems only by observing animals. Yet looks are often deceiving. Many birds that form pair bonds are not as loyal to one another as they may appear. When scientists analyzed the DNA of the chicks in nests of pair-bonding birds, they often found that a large fraction of the eggs did not carry the DNA of their mother's partner. Their mothers were mating with other males, and their partners were doing the hard work of raising the chicks.

To make sense of these intricate mating systems, evolutionary biologists consider how different strategies boost the reproductive success of males and females. The low cost of sperm, for example, can make polygyny a good strategy for males because they can fertilize many females, which can bear many offspring.

Polygyny has its downsides, though. Each male has to compete with lots of other males, and even if he succeeds in mating with females, the females may end up using sperm from other males to fertilize their eggs.

Under some conditions, a male may actually be better off mating with a single female, trying to fight off other males, and helping to raise the offspring. While most male mammals are not monogamous, for example, male California mice are (<u>Ribble 1991</u>). If a male California mouse helps rear his pups, more than twice as many of them will survive than if the mother rears them alone (<u>Gubernick and Teferi 2000</u>).

Polyandry can benefit females in several ways. By mating with a number of males, females may be able to get the highest-quality genes possible for their offspring. In essence, they "hedge their bets," in case sperm from any one of the males performs poorly. Each female superb fairy-wren forms a bond with one male, for example, but then slips off at night to mate with other males (FIGURE 9.14). The males they select on these forays show signs of being in particularly good condition, suggesting that these females are "upgrading" the quality of the sperm they use to conceive their chicks (Double and Cockburn 2000).



Top to bottom: Houshmand Rabbani/ Shutterstock; Katarina Christenson/Shutterstock.

FIGURE 9.14

Female superb fairy-wrens (*top*) form long-term bonds with males, but sneak off to mate with other male wrens.

Sperm Wars

Because females often mate with more than one male, they can end up with sperm from many males inside them at once. The stage is set for a new kind of competition between males: sperm competition. Various strategies have evolved that make some sperm more successful than others in fertilizing eggs. Not surprisingly, the strategies tend to be more common in species whose males compete more intensely for females—a pattern that you'd expect, given that strong competition leads to strong selection.

One way males can increase their reproductive success is by getting rid of the sperm females may be carrying from previous matings (FIGURE 9.15). Seed beetles (*Callosobruchus maculatus*) have sharp spines on their penises. When a male pairs with a female that has already mated with another male, the spines on his penis enable him to remove sperm from the earlier male. The longer the spines, the more success the male seed beetles have at fertilizing eggs (Hotzy and Arnqvist 2009).

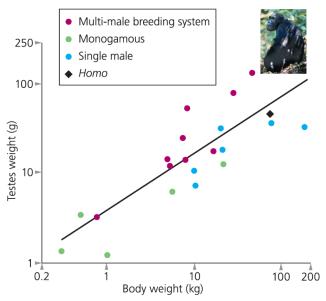


Doug Lemke/Shutterstock.

FIGURE 9.15

Male damselflies have sharp ridges on their sex organs, which they use to scrape sperm from other males out of female damselflies before they mate.

In some species, males have sperm that can outcompete sperm from other males also present in a female's reproductive tract. One such strategy is simply to swamp the opposition with sperm. Among primates, for example, the species with strong male—male competition have bigger testicles than do the species in which males and females tend to mate monogamously. Bigger testicles supply more sperm, and more sperm can raise a male's odds of fertilizing a female's eggs when sperm from other males are present (FIGURE 9.16).



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FIGURE 9.16

In primates, male testes are proportionately larger in species whose females mate with multiple males—that is, in species experiencing sperm competition. (Data from <u>Harcourt et al. 1981</u>.)

In deer mice (*Peromyscus*), a male's sperm will join together to form aggregates that can swim faster together than they can individually. Heidi Fisher and Hopi Hoekstra of Harvard, who discovered this cooperation, also found that the sperm can recognize other sperm from the same male, so that they preferentially aggregate with their kin. The researchers also discovered that males of a promiscuous species (*P. maniculatus*) with a history of strong

sperm competition were more likely to form aggregates with other sperm from the same male than were sperm from a monogamous mouse species (*P. polionotus*), which formed aggregates indiscriminately (<u>Fisher and Hoekstra 2010</u>).

After a male rat mates with a female, he inserts a plug of mucus into her reproductive tract. This copulatory plug is yet another strategy that may make it harder for other males to subsequently mate with that female. Steven Ramm, a biologist at the University of Liverpool in England, and his colleagues found that in species of rodents where competition between males is high, the male rodents tend to make larger plugs (Ramm, Parker, and Stockley 2005).

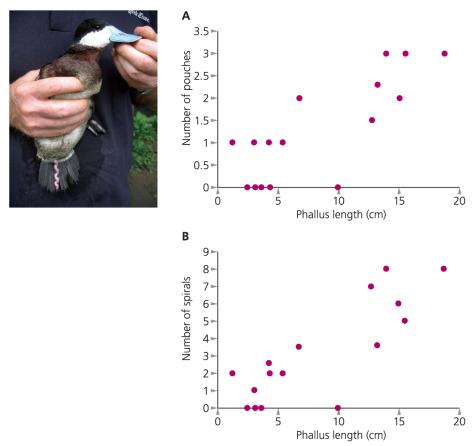
Sexual Conflict

Faster sperm, manipulative semen, mucus plugs, and other adaptations evolve as a result of males competing with one another for reproductive success. But while these adaptations may raise the fitness of a male, some of them also lower the reproductive success of females. As a result, females in many species have defenses of their own—defenses that can lower the fitness of males. This evolutionary conflict of interests between males and females is known as sexual conflict (<u>Arnqvist and Rowe 2005</u>).

Patricia Brennan's research on ducks and other waterfowl suggests that sexual conflict is driving the evolution of their bizarre sex organs. In many of these species, the males and females bond for an entire breeding season. Males that fail to find a mate of their own at the start of a season often harass females, trying to force them to mate. And when a bonded male's partner is busy incubating his eggs, he may also search for other females and force them to mate with him. All told, about a third of all matings are forced in some species of ducks and other waterfowl. The harassment from male ducks can get so intense that some females die as a result.

Although forced matings are common in ducks, the unwanted males father only about 3 percent of the ducklings each year. Brennan suspects that the female birds are controlling the sperm in their bodies, favoring the sperm from their partners over that of the intruders. One possibility she is investigating is that female ducks are shunting sperm into the side pockets in their oviducts.

Just like the evolution of measures and countermeasures that enhance survival in the face of parasites, sexual selection favors the evolution of measures and countermeasures that enhance reproductive fitness. Because males and females benefit from different strategies, when defenses evolve in females, sexual selection favors countermeasures from the males. In the case of ducks, longer, more flexible phalluses may evolve in males; and in response, even more twisted oviducts with more side pockets may evolve in females. Sexual conflict can explain why male and female sexual organs are so twisted in the birds Brennan has studied (FIGURE 9.17).



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: Bernard J. Brennan.

Male ducks, such as the one shown here, have long, coiled phallus. A: Patricia Brennan compared male and female sexual organs in ducks and other wading birds. Species with males that had long phalluses also had females with many pouches. B: Brennan and her colleagues also found that the length of the phallus in a bird species correlated with the number of spirals in the female reproductive tract. Both of these findings indicate that duck genitalia are the product of sexual conflict. (Graph data from Brennan et al. 2007.)

Fitness for a Lifetime

From stalk-eyed flies to elephant seals, animals have evolved a stupendous variety of mating strategies. But the shaping force of evolution does not stop when an egg is fertilized. After all, an organism's fitness depends on the total number of offspring that it can produce over its lifetime—as well as on how many of those offspring survive to adulthood. If certain genes allow an organism to increase its lifetime fitness, those genes will be favored by selection.

This insight—first hit upon by the American evolutionary biologist George Williams (Williams 1966)—has helped scientists make sense of many features of biology that might otherwise be a mystery. Why is it that some species live for a long time and others are short lived? Why do some species spend years raising a few young while others abandon their eggs? In many cases, scientists have found an explanation in the way evolution acts over a lifetime.

Many factors go into the lifetime fitness of an organism, and they can come into conflict (FIGURE 9.17). If a bird has genes that allow it to lay extra eggs in each clutch, for example, it potentially can have more reproductive success than other female birds. But once those eggs hatch, the bird has to provide enough food for its chicks so that they can reach maturity. A bird that can find enough food for four chicks won't be able to find enough for 40. As a result, many of her offspring may die, wiping out the evolutionary benefit of a large clutch of eggs.

Organisms also face trade-offs between youth and old age. In many species, individuals that can develop slowly over long periods can reach large sizes and enjoy greater health through their mature years. On the other hand, an organism that reaches sexual maturity quickly can start reproducing sooner, although it may suffer poor health later in life.

Williams argued that selection should favor a balance between an organism's traits that maximized the number of offspring it could successfully rear to maturity. That balance depends on the particular biology of a given species as well as the environment in which it evolves. That environment

may have a lot of predators in it, for example, or just a few. Organisms that face many predators will tend to die young. In those populations, selection will favor mutations that improved their survival and reproduction at young ages. Mutations that have harmful effects at advanced age won't be eliminated by selection, because most organisms already have reproduced and then died before those mutations could have any effect.



Stephen J Krasemann/All Canada Photos.

FIGURE 9.18

Opossums give birth to seven or eight babies in a single litter, nurturing them with milk until they can live on their own. How many offspring they have and how much they invest in their rearing are traits shaped by natural selection.

This trade-off, Williams and other scientists realized, could even explain the life span of species. In the late 1970s, Thomas Kirkwood, an evolutionary biologist at Newcastle University in England, noted that organisms of any age are continually faced with damage to their cells (Kirkwood and Holliday 1979). To survive, they have to fix DNA replication errors, replace deformed proteins with new copies, and produce

molecules that can shield cells from damage. Kirkwood argued that natural selection should favor just enough self-repair to keep an organism in sound condition only for as long as it has a reasonable chance of reproducing.

More than 90 percent of mice in the wild die in their first year, for example. According to Kirkwood's theory, a mouse that invests in mechanisms for survival beyond its first year has only a 10 percent chance of experiencing any benefit from its self-repair. So mice that invest enough energy in repair to keep their cells in good working order for a decade should be outcompeted by mice that invest that extra energy into producing lots of pups in their first year.

In the 1980s, Steven Austad, who now teaches at the University of Texas Health Science Center in San Antonio, became curious about these ideas when he studied opossums in South America and New Guinea. He found that once opossums reached about 18 months of age, their health declined rapidly. They quickly developed cataracts, arthritis, and other symptoms of old age. Most were dead within two years.

If Williams and Kirkwood were right, then this rapid decline should be the result of the mortality faced by opossums. If Austad could find populations of opossums with different mortality rates, he suspected he might find different patterns of aging.

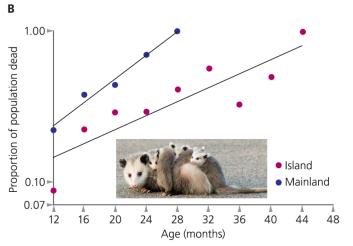
Austad discovered just such a natural experiment in the southeastern United States. On the mainland, 80 percent of opossums are killed by predators. But on Sapelo Island, just off the coast of Georgia, there are opossums but no predators. During the Ice Age, the island had been joined to the mainland; but when the glaciers melted, sea levels rose. The island, and its opossums, became isolated. For the past five thousand years, opossums living there have been free of predation.

The Sapelo Island opossums, Austad discovered, lived 25 percent longer than their mainland cousins. It was as if he had discovered an isolated tribe of people who regularly lived well past 100 years (<u>Austad 1993</u>). That sort of difference is what you'd predict from the theories of Williams and Kirkwood: the high mortality among mainland opossums favored mutations that ended up shortening their lives.

To investigate the mechanisms underlying this difference in life span, Austad studied how the animals repaired their cells. In particular, he looked at the collagen in the opossums. Tendons are made of collagen fibers, which slide past each other to allow the tendons to stretch. As animals get older, the fibers form links and become stiffer. Austad found that the tendons in the island opossums had fewer links than did those of mainland opossums of the same age. In other words, the island opossums were aging more slowly.

The Sapelo Island opossums produced fewer offspring in each litter than mainland females did, Austad found. The island opossums were more likely to survive to their second year and deliver a second litter, and so they no longer had to invest as much energy into their first. Because more opossums on Sapelo Island survived longer, a slower—yet successful—reproductive strategy evolved (FIGURE 9.19).

А			
Comparison between Sapelo and mainland opossums			
	Mainland	Island	P value
Longevity (mo)	20.0	24.5	0.002
Litter size (1st year)	7.6	5.9	0.002



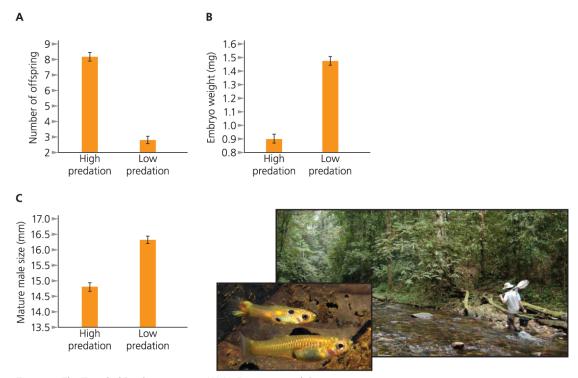
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FIGURE 9.19

Steven Austad compared opossums living on Sapelo Island off the coast of Georgia, where there are no predators, to their mainland counterparts. A: He found differences in the timing of their life history. B: The death rate increased more slowly on the island than the mainland. Such results are consistent with the hypothesis that the absence of predators influenced the evolution of the opossums on the island.

More support for such trade-offs has come from research on guppies (*Poecilia reticulata*) that live in the streams of the Caribbean island of Trinidad. The eggs in this species hatch inside their mothers and are born live. The resources a female guppy gives an offspring will influence its odds of surviving until adulthood (<u>Reznick 2011</u>). David Reznick, an evolutionary biologist at the University of California–Riverside, and his colleagues compared the guppies in streams with predators to those in predator-free waters.

The scientists found some differences between the two kinds of guppies. Predator-free guppies grow up slowly, and the females produce large offspring—but few offspring per litter. In the guppy populations that are menaced by predators, on the other hand, a different strategy has evolved. The males are ready to mate much earlier than the males at predator-free sites. The females produce twice as many offspring, but each baby is only 60 percent as large as the average baby produced by females in predator-free sites (FIGURE 9.20).



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Left to right: Paul Bentzen; Andrew Hendry.

David Reznick and his colleagues compared guppies in streams with many predators to those in predator-free waters. As these graphs show, the guppies that faced few predators produced fewer offspring (A), and bigger offspring (B), than the guppies menaced by many predators. The guppies also grew to larger sizes by the time they were sexually mature (C). Natural selection shaped the life history of the guppies in different ways, depending on whether they were more or less likely to be killed by predators. (Data from Reznick et al. 2006.)

Reznick and his colleagues argue that these patterns are the result of the trade-offs Williams and Kirkwood have proposed. To test this hypothesis, they ran an experiment. They moved some of the guppies that lived with predators over to predator-free streams. Over the course of 11 years, the fishes adapted to the new environment.

The scientists then collected some guppies from the predator-free transplant site and the original predator-dense site. They brought the fishes to their laboratory, where the guppies reproduced under identical conditions. In the absence of predators, Reznick and his colleagues found, female guppies evolved to produce smaller broods, and their offspring took longer to reach maturity. The fishes grew to be bigger than their counterparts that still lived with predators.

Reznick's experiment illustrates just how quickly populations can respond to selection for life-history trade-offs. It's only a matter of years—not millennia.

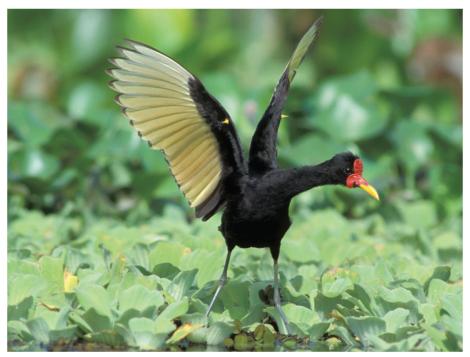
Trading Places

One very striking pattern of parental care is its huge imbalance between the sexes. In most species, females provide most of it—or even all of it. This imbalance emerges from the limits to reproductive success that we examined earlier in this chapter. Since the fitness of females is limited by the number of eggs they can rear, they can benefit greatly from caring for their young. The cost of feeding and protecting their young will be outweighed by the increased odds that their offspring survive long enough to reproduce themselves. For most males, the cost-benefit equation is different. A male producing many cheap sperm invests less to begin with, and he usually loses less if he elects to abandon the young.

Females can also be more certain that the care they invest in their offspring is actually going to pay off in an evolutionary sense. When a female gives birth, there can be no doubt that her offspring developed from her own eggs. Males, on the other hand, have a much harder time recognizing their offspring, because females may mate with several different males. A male that invests a lot of effort raising offspring that may or may not be his own risks wasting time that could be spent finding other mates.

Both the limits on reproductive success and the different levels of certainty have led to females providing most of the parental care in most species. The pattern is not universal, however. In a few animal species, for example, the male is the one that puts in the hard work of rearing young. But these exceptions prove the rule.

Among wattled jacanas (*Jacana jacana*), for example, a female lays her eggs in a male's nest and then abandons them (FIGURE 9.21). The male jacana spends a month with their brood, first protecting the eggs and then providing for the chicks after they hatch. Female jacanas fight with rival females for the possession of territories that include male nests, and they mate with all the males they control. The female jacanas are larger than males, and they are more aggressive—on their wings are spurs that they use as formidable weapons to fight with rivals (Emlen and Wrege 2004).



Marie Read/Science Source.

Among wattled jacanas, the typical sexual roles are reversed. Males rear eggs in nests, and females guard harems of multiple males. Females (like the one shown here) are larger, and they have larger weapons (yellow wing spurs) than males.

In some species, parental role reversal becomes so extreme that the males effectively become pregnant. Such is the case among sea horses and their relatives, known collectively as syngnathid fishes. Among these species, it's the males that get pregnant (FIGURE 9.22). When syngnathid fishes mate, the female transfers her unfertilized eggs to the male. The male stores the eggs, sometimes inside a fleshy pouch, where he fertilizes them with his sperm. While sperm in other animals may be champion swimmers that can make the long journey up a female's reproductive tract, syngnathid sperm barely move at all.



NaturePL/Superstock, Inc.

In sea horses and related species of fishes, males become pregnant. They carry developing eggs, which they provision with nutrients.

As the eggs develop in males, some of their energy comes from the yolk provided by their mother, and some energy comes from their fathers. The pouch of some species changes shape, taking on a complex anatomy that brings each fish embryo in intimate contact with the father's blood supply, so that he can give them nutrients. Eventually the baby fish wiggle out and are ready for life on their own.

In this mating system, the females still produce the eggs, but they don't have to put the time and effort into rearing them. Sexual selection theory suggests that they would be better off looking for as many males as possible to take their eggs. And with all those females swimming around in search of males, they're going to face some fierce competition.

Adam Jones of Texas A&M University and his colleagues have studied a syngnathid fish called gulf pipefish to see whether this reversed competition in fact occurs. In one experiment, they found that most females failed to find any mates at all, but a small proportion of the females managed to mate with

four males. Most of the male pipefish, on the other hand, mated once during Jones's experiment (<u>Jones, Walker, and Avise 2001</u>).

Male syngnathid fishes are not limited by the number of females they can fertilize. As a result, they don't benefit from making lots of sperm. And that explains the remarkably scant supply of sperm these fish produce. Instead of making millions of sperm every day, a male sea horse's testes may carry just 150 sperm in total.

The competition of females opens up the opportunity for males, rather than females, to be picky. Jones and other scientists found that, indeed, the females that mate the most have certain traits in common. They tend to be bigger than other females, and they have fancier fins and brighter colors. Jones found that big females transfer more eggs into the pouches of males than small females. And the bigger the female, the more likely each egg is to survive. The preferences of the males cause large females to have more offspring.

Jones and his colleagues also wondered if males controlled the amount of investment they put into rearing eggs from different females. The researchers had males mate with one female and then another. They discovered that the survival of the second brood depended on the first. If the first brood came from a big female, fewer of the eggs from the second brood survived. Jones and his colleagues concluded that the males were likely giving more resources to the eggs from big females, leaving less for small females they might later encounter. And if they did end up mating with smaller females, they should give the eggs fewer resources, storing more for themselves so that they're in a better position should they encounter a big female next time around (Paczolt and Jones 2010).

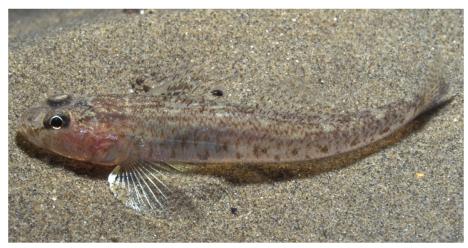
Adjusting the Family

Investments can be risky. If you put a thousand dollars into a promising startup company, that money may multiply many times over, or it may vanish. Parents face a similar uncertainty when they invest resources in their offspring. Mutations can arise, causing birth defects. A female may carry a fetus for months, only to produce an offspring that ultimately will be unable to reproduce. Changes to the environment can also bring reproductive risks. In some years, a female bird may be able to provide large amounts of food to her chicks thanks to abundant rain and a bumper crop of seed-bearing plants. In other years, a drought may leave her struggling to find enough food to keep them alive.

Evolutionary theory suggests that natural selection should favor adaptations that reduce these risks in order to raise fitness over an entire lifetime. One way to cope with the uncertainty of reproduction is to use a flexible strategy to respond to different challenges. In many species, parents use just these kinds of strategies. In some cases, they can adjust the number of their offspring to raise their long-term fitness; in other cases, they can manipulate the ratio of sons to daughters in their offspring.

Miscarriages are sometimes the result of these flexible strategies. When fertilized eggs begin to develop, they may carry harmful mutations. In some cases, for example, they have extra copies of chromosomes, leading to Down syndrome and other disorders. Scientists have found that 90 percent of human embryos with abnormal chromosome numbers result in miscarriages in the first trimester of pregnancy. On the other hand, 93 percent of embryos with normal chromosome number continue to term (Quenby et al. 2002). A number of scientists have argued that these miscarriages are not simply the failure of an embryo to develop. Instead, the mother's body may be using chemical cues to assess offspring quality and spontaneously abort embryos that show signs of genetic abnormalities (Forbes 1997). Miscarriages are a source of great psychological suffering, but they are favored by natural selection because they can reduce nine months of investment to a month or less.

The sand goby (*Pomatoschistus minutus*), a European species of fish, uses a different strategy to cut its losses: cannibalism (<u>FIGURE 9.23</u>).



Paul Kay/Getty Images.

FIGURE 9.23

Male sand gobies care for nests of eggs. If conditions deteriorate, the fathers will cannibalize some of the eggs to boost the survival of the remaining offspring.

To rear its young, a male sand goby builds a nest in an empty shell. After successful courtship, a female goby releases her eggs, which are then fertilized by the male. The male sand goby then tends the nest, covering it with sand, guarding it from predators, and cleaning off any algae. Sand goby fathers even fan the eggs to provide them with a fresh supply of water as they develop.

Male sand gobies will sometimes do something that seems unthinkable: they dig up their nests and devour their own eggs. Hope Klug, a biologist at the University of Helsinki, found an evolutionary logic behind this cannibalism (Klug, Lindströum, and Mary 2006). She experimentally raised and lowered levels of oxygen in the nests. The number of eggs the father ate depended on how low the oxygen levels dropped.

Klug concluded that by selectively eating some of the eggs, the fathers were increasing the survivorship of the remaining eggs. The denser the eggs in a nest, the more eggs the father was likely to eat. Rather than being a random act of destruction, the cannibalism appears to be an adaptive

response the males make to certain changes in their environment—cannibalism results in increased egg survivorship.

In addition to adjusting the number of developing offspring, animals can maximize their fitness by adjusting the ratio of sons and daughters away from the typical one-to-one proportions. Ronald Fisher offered an elegant argument for why this balance is normally in place (Fisher 1930). Imagine that mutations lead to more female births than male births. The imbalance gives males an advantage; a male is more likely to find a mate than a female. If mutations arise that make some animals produce more sons, they will be favored by natural selection. But, as the numbers of males come to equal the numbers of females in each new generation, the advantage of being male dwindles. The same process would work under the opposite conditions, when there are more males than females. The sex ratio of the population balances itself at one to one, in an example of frequency-dependent natural selection.

In 1973, Robert Trivers and Dan Willard, both then at Harvard, argued that natural selection could drive sex ratios away from one to one under certain conditions. Consider, for example, a species in which a few males in good condition mate with most of the females. If a female is in good condition herself, she may be able to boost her reproductive success by having more sons than daughters. Her sons, in good condition themselves, will mate with many partners and give her more grandchildren than daughters would.

On the other hand, if a female is in poor condition, Trivers and Willard argued, she may be better off having more daughters than sons. Sons in poor condition may fail to attract any mates at all, and may therefore leave their mother without any grandchildren. Daughters, on the other hand, will probably have at least some offspring, even if they are in poor condition.

Trivers and Willard proposed that mothers could switch the sex ratio of their offspring to suit their condition. Such is the case for the Seychelles warbler, a bird that lives on the Seychelles Islands in the Indian Ocean. When a mother warbler's eggs hatch, the male and female chicks can look forward to different lives. The male birds tend to fly away from their natal home in search of other female warblers and other territories. Young female birds tend to stay behind, helping their mother incubate her eggs. A mother benefits from this help because she is able to rear more chicks over her

lifetime with the aid of her daughters (<u>Brouwer, Richardson, and Komdeur 2012</u>).

Jan Komdeur, a biologist at the University of Groningen in the Netherlands, and his colleagues have been carefully chronicling the lives of all two thousand or so Seychelles warblers that live on the islands. They discovered that female Seychelles warblers can adjust the balance of male and female chicks in their broods in response to their environment. An unassisted female living on a patch of land with abundant food may produce a brood that's as much as 88 percent daughters. But Komdeur has found that female warblers that live in places where food is scarce may produce broods in which as few as 23 percent of the chicks are daughters (FIGURE 9.24).



Rene van Bakel/ASAblanca/Getty Images.

FIGURE 9.24

Female Seychelles warblers can adjust the ratio of sons to daughters to maximize their reproductive success.

Komdeur hypothesized that the birds were adjusting the balance of daughters and sons to maximize their reproductive success. A female bird that lives on a high-quality territory can use the help of her daughters to produce more chicks. A female that is stuck living on a low-quality territory will be better off producing sons that can search for greener pastures. Komdeur tested his hypothesis by moving birds from low-quality territories to high-quality ones. And as he predicted, the birds switched from mostly sons to mostly daughters.

There is, however, such a thing as too much help. When Seychelles warbler mothers living on high-quality territory have more than three female helpers, trouble arises. The helpers eat too much food, and they may crack the mother's eggs as they clamber around the nest. In response, the Seychelles warblers adjust the sex ratio yet again. They produce more sons that will soon fly away and not be such a burden. To test his hypothesis in a new way, Komdeur took away the helpers in some of the warbler nests. The sex ratio changed again, and in exactly the way he predicted: the unassisted birds started producing more daughters again.

Menopause: Why Do Women Stop Having Children?

Human sexuality and parenting are shaped by evolution, as they are in other species. One of the most intriguing features of our biology is menopause. After about age 50, women stop reproducing. This pattern is strikingly different from that of our closest relatives. Female chimpanzees also experience a decline in their fertility, but only at the end of their lives, when their entire bodies are in decline. The oldest confirmed chimpanzee lived for 59.4 years. Women, by contrast, may live for many decades past their reproductive years.

Menopause is not the result of better hygiene, nutrition, or medicine made possible by modern civilization. Women in hunter-gatherer societies experience menopause as reliably as women living in affluent cities. The universality of menopause strongly suggests that it is a biological feature of our species—one that evolved at some point after our ancestors branched off from other apes some 7 million years ago.

Evolutionary biologists have proposed several different hypotheses to explain how women's reproduction changes over their lifetime (Kirkwood and Shanley 2010). In 1957, George Williams first proposed what came to be one of the most influential of these hypotheses. Williams pointed out that a woman in her late 40s would have a harder time caring for a newborn than she would when in her 20s. What's more, she would have to divide her limited resources between rearing babies and continuing to care for her older children. Williams suggested that natural selection favored mutations that reduced a woman's fertility so that she could focus on raising her older children instead (Williams 1957). Other researchers have elaborated on the so-called mother hypothesis (Pavard, Metcalf, and Heyer 2008). They argued that older women who became pregnant faced a much more direct threat to their reproductive success: they were more likely to die in childbirth, leaving their other children at grave risk of dying as well.

But other researchers have tested the mother hypothesis and found it wanting. Virpi Luumaa of the University of Sheffield and her colleagues analyzed historical records from Canada and Finland to measure the costs older women pay for childbearing. The records from Finland covered the years from 1741 to 1908, and those from Canada stretched from the nineteenth century into the twentieth. The mothers covered in these records generally lived on farms without access to modern medicine. Luumaa and her colleagues found that the risk of death from childbirth was real, though small. At age 50, women faced only a 1–2 percent risk of dying during labor. And even when women did die, the effect on their surviving children was also small. Only children who were not yet weaned faced an increased risk of death if their mothers died in giving birth to another child. Because humans have traditionally lived in extended families, the scientists concluded, children who lose their mother can get sufficient care from other family members (Lahdenperä et al. 2011).

Luumaa and her colleagues suggested a variant on the mother hypothesis—known as the grandmother hypothesis—that may explain prolonged life span, but not necessarily menopause (FIGURE 9.25). For most of human history, many of the women who were still alive in their late 40s had become grandmothers. By helping their children raise their grandchildren, older women could raise the odds that their genes would be passed down to future generations. In their studies of historical records, Luumaa and her colleagues found that children were more likely to survive till adulthood if their grandmothers were still alive (Lahdenperä et al. 2004).



Library of Congress.

Human females pose an evolutionary puzzle: why do they experience menopause? According to one hypothesis, shifting care to grandchildren provides more reproductive success than trying to raise more children.

Both the mother hypothesis and the grandmother hypothesis present menopause as an adaptation—a trait that has evolved through natural selection because it has a function that raises reproductive success. But some scientists dispute this idea. Caleb Finch of the University of Southern California and Donna Holmes of Washington State University observe that the life histories of human and nonhuman females have some similarities that have gone unappreciated (Finch and Holmes 2010). In many species of vertebrates, females typically set aside a population of eggs when they are still embryos. As time passes, these eggs gradually become damaged. Eggs have more self-repair mechanisms than typical cells, but nevertheless they become less viable over time. Thus the mechanism that leads to infertility in menopausal women is the same in other animal species. Finch and Holmes also observe that females in other species, including elephants and rats, have sometimes survived for a relatively long time after losing their ability to reproduce.

Even guppies have a form of menopause. Holmes, Reznick, and Reznick's student Michael Bryant found that in female guppies in Trinidad, the median post-reproductive life span was 13.6 percent of total life span (Reznick, Bryant, and Holmes 2006). To see if this guppy version of menopause was subject to selection, they compared the life histories of guppies from high-and low-predation environments. While the two populations of guppies exhibited significant differences in every other major feature of life history, there was no difference when it came to the length of time females lived after they stopped reproducing. This study suggests that rather than being an adaptation that evolved through direct natural selection, menopause may simply be another result of aging's trade-offs.

The debate over menopause is far from over. It will fuel new research on human and nonhuman life-history patterns. And, like other research into sex and parenting, the debate will rely on the evolutionary framework we've explored in this chapter.

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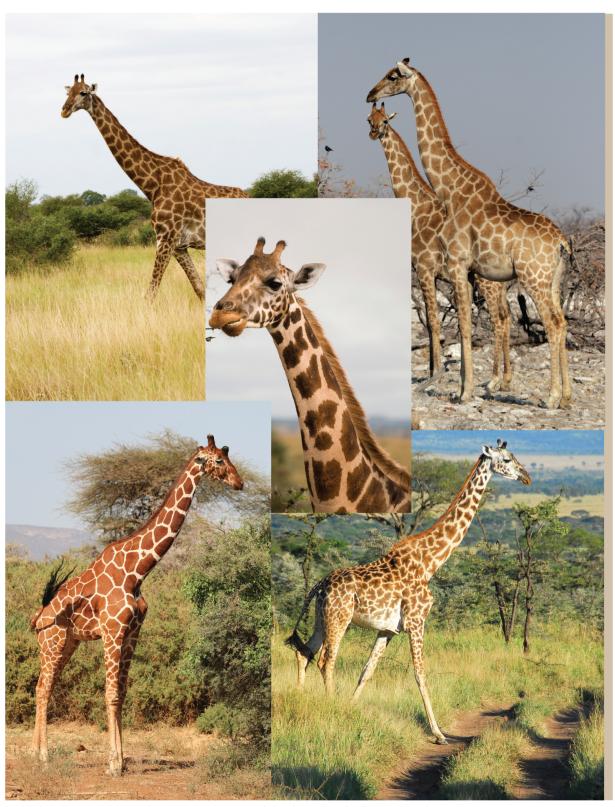
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Darwin's First Question

10

The Origin of Species



(top left to bottom right) Richmatts/Getty Images; dirkr/Shutterstock; Klaas Lingbeekvan Kranen/Getty Images; Liz Leyden/Getty Images; Brian Raisbeck/Getty Images.

Living giraffes have traditionally been classified as a single species. However, recent studies suggest there may be six species, each living in a different range in Africa.

Rick Brenneman loaded his rifle and quietly took aim. He had come thousands of kilometers from his home in Omaha, Nebraska, to stalk giraffes on the Hoanib River, which flows through a remote corner of Namibia in southern Africa. He selected a tall bull from a herd and fired a direct hit. But the bull giraffe did not collapse and die. Instead, it staggered a little and then ran away. There were no bullets in Brenneman's rifle; instead, he shot a needle into the giraffe's side. The needle then fell out with a tiny bit of skin lodged in its tip. Brenneman and his colleagues walked across the savanna and picked up the needle. From the bit of skin it held, Brenneman would extract a trophy far more valuable to him than a giraffe's skin: giraffe DNA.

Brenneman is a conservation geneticist at the Henry Doorly Zoo in Omaha. He and his colleagues have been firing darts at giraffes across Africa to help save them from extinction. Although giraffes live throughout much of Africa, their population has dropped by 30 percent in just the past decade, leaving fewer than 100,000 individuals in the wild. Brenneman and his colleagues are analyzing giraffe DNA to learn more about their biology so that they can develop well-informed conservation plans.

You might think there isn't much left to learn about giraffes. After all, their tall necks tower over the African plains, making them an

easy animal to track. They don't lurk among the microbes hidden in the soil or in some deep underwater canyon. And yet Brenneman and his colleagues discovered something quite astonishing when they compared the DNA of 266 giraffes from Namibia, Kenya, Niger, Uganda, Zimbabwe, Tanzania, and South Africa. If you look in a standard field guide to African wildlife, you'll find a single species of giraffe on Earth: *Giraffa camelopardalis*. But after Brenneman and his colleagues analyzed the DNA, they concluded that there are six species of giraffes—not one.

How could six species of giraffes be hiding in plain sight? That's one of the questions we'll try to answer in this chapter. But first we'll have to examine the meaning of the word *species*. We will examine the mechanisms that keep species distinct from one another today, and then we will turn to how those mechanisms arise, splitting new species off of old ones. As we'll see, many factors can come into play in the evolution of a new species, including natural selection, sexual conflict, and genetic drift.

Understanding speciation is important for many reasons. It's crucial for doctors who need to recognize the species of pathogens that infect their patients. It's also vital for conservation biologists who strive to preserve the world's biodiversity. There's a big difference, after all, between how you conserve one species of giraffe and how you conserve six.

What Is a Species?

Long before the dawn of science, humans were naming species. To be able to hunt animals and gather plants, people had to know what they were talking about. Taxonomy, the modern science of naming and classifying species, emerged in the 1600s and came into its own in the next century, thanks largely to the work of Swedish naturalist Carl Linnaeus.

As we saw in <u>Chapter 3</u>, Linnaeus invented a system to sort living things into groups, inside which were smaller groups. Every member of a particular group shared certain key traits. Humans belonged to the mammal class, and within that class the primate order, and within that order the genus *Homo*, and within that genus the species *Homo sapiens*. Linnaeus declared that each species had existed since creation. "There are as many species as the Infinite Being produced diverse forms in the beginning," he wrote (<u>Wilkins 2009</u>).

Linnaeus's new order made the work of taxonomists much easier. But trying to draw the lines between species often proved frustrating. Two species of mice might interbreed where their ranges overlapped, raising the question of what name to give the hybrids. Within a species there was confusion as well. The willow ptarmigans in Ireland, for example, have a slightly different plumage than the willow ptarmigans in Finland, which differ in turn from the ones in Norway. Naturalists could not agree about whether the birds belonged to different ptarmigan species or were just varieties of a single species.

Charles Darwin, for one, was amused by these struggles. "It is really laughable to see what different ideas are prominent in various naturalists' minds, when they speak of 'species,'" he wrote in 1856. "It all comes, I believe, from trying to define the indefinable."

To Darwin, naturalists of his day were hobbled by the notion that species were fixed since creation. Darwin argued instead that species had evolved. Each group of organisms that we call a species starts out as a variety of an older species. Over time, forces such as natural selection transform them independently in their separate environments. When these varieties have diverged sufficiently from each other, we see them as distinct species in their

own right. In this way, new species arise over time, just as others become extinct.

"I look at the term 'species' as one arbitrarily given, for the sake of convenience, to a set of individuals closely resembling each other," Darwin declared.

Once Darwin published *The Origin of Species*, other biologists came to accept that species were the products of evolution. But they still needed a way to talk about species—a concept to guide their research. Over the years, they've come up with many alternatives, each influenced by the aspect of evolution they study. Biologists who use DNA to reconstruct phylogeny need a concept that allows them to recognize a species from genetic information, for example. Many of them favor a so-called phylogenetic species concept, in which a species is a "tip" of a phylogenetic tree.

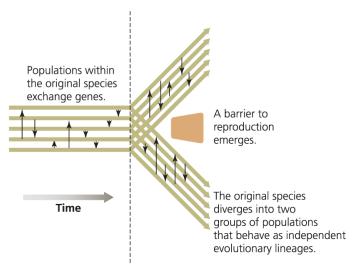
The ornithologist Ernst Mayr defined a species in a different way. He called it a group of "actually or potentially interbreeding populations which are reproductively isolated from other such groups" (Mayr 1942). Scientists now refer to this as the biological species concept. It has proved enormously useful because it focused on the process of how species form. Other species concepts tended to emphasize how you recognize a species once it is there. Mayr's was all about the things that might keep populations apart.

But the biological species concept has its drawbacks. It can be applied only to organisms that reproduce sexually. It implies that the only way to determine whether a species is real is to observe whether it interbreeds with other species—which is a daunting task in many cases. And in a number of cases, interbreeding does not eliminate the morphological differences between species. In Europe, 16 percent of all butterfly species can interbreed and produce viable hybrids (Mallet 2008). That interbreeding should arguably disqualify them from being species under the biological species concept. And yet you can easily distinguish them with the naked eye.

Today, biologists generally agree that no single concept will ever fit all of life (<u>Coyne and Orr 2004</u>). Rather than try to chase after such a concept, it's more rewarding to explore the process by which species evolve and are maintained.

Keeping Species Apart

There are trillions of animals and plants on Earth, and at any moment a vast number of them are mating or producing offspring. But they do not all mate with each other at random. They're divided by barriers of many forms (FIGURE 10.2). Geography creates many of the most effective barriers between species (this geographical isolation is called allopatry). Elk in North America and red deer in Europe and Russia are very closely related, for example, but their ranges are separated by thousands of miles of ocean (FIGURE 10.3).



Zimmer, The Tangled Bank, 2e, © 2014 W. H. Freeman and Company

FIGURE 10.2

A species divides in two as barriers prevent its populations from interbreeding. The barriers can be either geographical or based in the biology of the organisms. (Information from Doolittle 2008.)



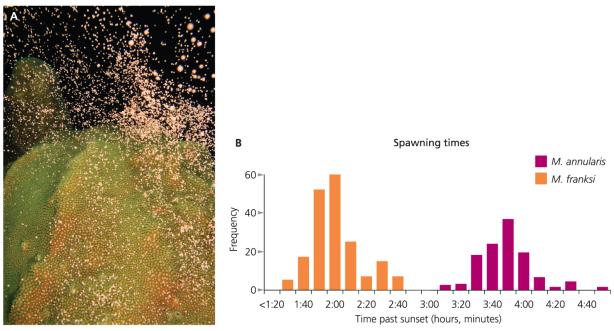
Top to bottom: Paul Tessier/ Getty Images; Damian Kuzdak/Getty Images.

Elk (top) and red deer are geographically isolated but closely related. Scientists debate whether they belong to a single species or two.

Does the allopatry of red deer and elk mean that they should be considered two separate species? Not necessarily. If you were to put an elk on a plane and fly it to Hawaii—a place where elk have never lived—it wouldn't make sense to declare it a new species simply because it was geographically isolated from other elk. After all, it could still interbreed with North American elk if you gave it the opportunity. Likewise, red deer and elk can interbreed when they live together in zoos. As a result, many scientists consider red deer and elk two populations of a single species, *Cervus elaphus*.

Species that share the same geographic area are said to live in sympatry. Since they can't be separated by geography, the barriers that prevent these species from mating must emerge from their biology. In some cases, the way they seek food helps keep sympatric species separate. In many species of fruit flies, for example, males and females converge on a particular species of fruit to find a mate. The females lay their eggs in the same fruit, so that when the eggs hatch they have a ready supply of food. The fruit flies that specialize on, say, hawthorns will rarely mate with fruit flies that specialize on blueberries, even if their range is identical (Feder, Chilcote, and Bush 1989).

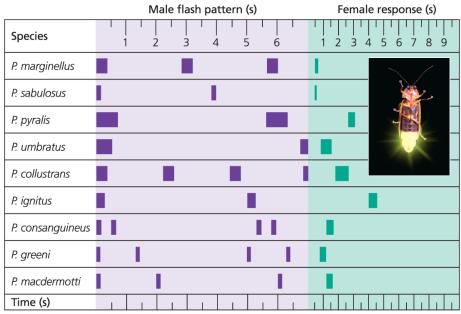
Time can also create reproductive barriers. Corals, for example, mate by spewing sperm and eggs into the water (FIGURE 10.4). When two gametes meet, they produce a fertilized egg. Each population of corals releases its gametes in a burst lasting 15 to 30 minutes. It doesn't take long after that for the gametes to be dispersed by the ocean currents. Scientists have found that sympatric coral species release their gametes at different times of day, minimizing the chances they'll interbreed (Levitan et al. 2004).



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A: Corals reproduce by releasing gametes into the water. B: This graph shows how two species of *Monostrea* corals remain reproductively isolated: by spawning at different times after sunset. (Data from <u>Levitan et al. 2004</u>.)

In the previous chapter, we saw how sexual selection has driven the evolution of elaborate courtship rituals in some animal species. Females will respond only to distinctive rituals—even a slight departure may leave them uninterested in mating. Fireflies use flashes of light for courtship, for example, and in a single field in Massachusetts, as many as six species of fireflies may carry out their courtships at the same time (FIGURE 10.5). The males of each species produce a distinctive series of flashes, and the females respond only to the ones they find attractive. What's more, the males approach only the females that wait a suitable and species-specific period of time before responding with a flash of their own. These signals and preferences create a reproductive barrier between these firefly species (Lewis and Cratsley 2008).



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FIGURE 10.5

Male fireflies of different species produce flashes in different patterns. Some flash many short pulses while others produce a few widely spaced flashes. Females respond only to

signals from males of their own species. Even when many firefly species live side by side, this flash behavior creates a barrier that helps prevent them from interbreeding. (Data from <u>Lewis and Cratsley 2008</u>.)

All of these barriers block reproduction before a sperm fertilizes an egg. But after conception, there are many further opportunities for more barriers to arise. To begin with, embryos with two species for parents may fail to develop. Hybrids do sometimes develop successfully; but some of them are born with deformities that leave them in poor health, and others are healthy but sterile. Mules, which are the hybrid offspring of horses and donkeys, are almost never able to breed with each other, nor can they breed with horses or donkeys. Thus the genes from the horses cannot flow into the donkey population, and vice versa. Even though mules are viable animals, their sterility means that horses and donkeys remain distinct species.

The Origin of Isolating Barriers

Reproductive barriers don't just keep existing species separate. They can also arise within a species and split it apart. It's been over 150 years since Darwin conceived of this process, known as speciation, but only recently have scientists begun to uncover the underlying genetic changes (Nosil and Schluter 2011).

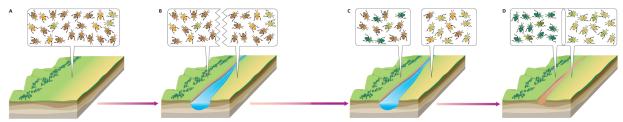
Biologists use the insights they get from such experiments to test hypotheses about how new species evolve in the wild. In the mid-1900s, as the science of population genetics was coming into its own, biologists offered the first detailed account of how reproductive barriers could evolve in nature. Ernst Mayr and Theodosius Dobzhansky, a geneticist who studied the fruit fly *Drosophila*, offered different versions of the same basic idea: new species evolve when old ones are geographically split. When populations become separated, the gene flow between them declines, and they begin to diverge.

Populations of a species can become geographically divided in many ways. A few birds are swept away by a storm to a distant island, for example. A glacier slices down through the range of a salamander, leaving isolated populations on either side. A species of butterfly that lives in the lowlands of a mountain range shifts uphill as the climate warms. Eventually the butterflies become isolated on the mountain peaks, their once continuous range transformed into a broken chain of islands in the sky.

The divided populations continue to evolve: new mutations arise in each one, and some of them become fixed through genetic drift. Each population accumulates its own unique set of mutations, and the longer they are divided, the more of these mutations they accumulate.

At the same time, natural selection also acts on the populations. If they face different conditions, natural selection favors different adaptations. Fruit flies swept to a new island may encounter new kinds of fruit, which they can break down with modified enzymes. In each population, males and females continue to adapt to one another through sexual selection and sexual conflict (Chapter 9). Over time, the populations may begin to differ in ways that

would cause them to be less compatible if they were later brought back into contact. That is, once populations have begun to evolve independently, reproductive barriers to gene flow can begin to accumulate (FIGURE 10.6).



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FIGURE 10.6

Allopatric speciation is the result of geographical isolation. A: Here, a population begins with a continuous geographical range. It contains genetic variation, but gene flow ensures that new mutations spread across the range. B: A river divides the population into two subpopulations. C: When the alleles in one subpopulation change, the other subpopulation does not acquire the same changes. They become increasingly divergent, and reproductive barriers evolve. D: The river later dries up, allowing the two subpopulations to make contact. If the barriers are strong enough, the two subpopulations still will not interbreed much, if at all.

Populations that are geographically divided sometimes come back into contact again. A glacier that divides a salamander species in two may melt. The descendants of birds that colonized an island may return to the mainland. A cooling climate may allow butterflies to return from their mountaintop refuges and encounter butterflies from other mountains. These populations will now have the opportunity to interbreed. What happens next depends on how strong the reproductive barriers have become.

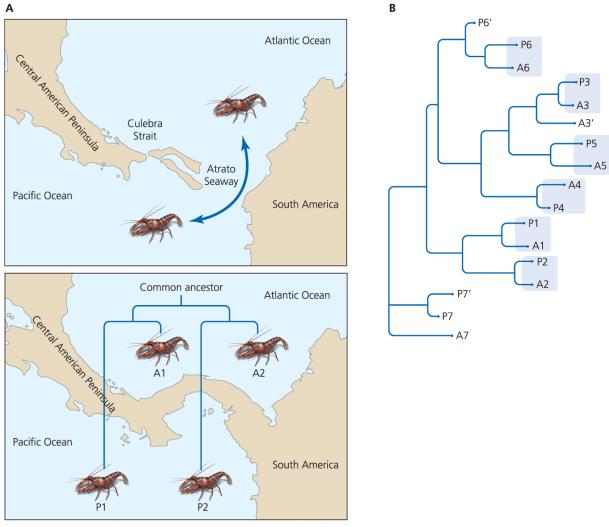
If the barriers are weak, the population will interbreed easily with the rest of its species, and any accumulated genetic differences will quickly disappear. If the reproductive barriers are very strong, the populations may not interbreed at all, and the process of speciation will be complete.

In other cases, the two populations may produce hybrids, but the hybrids have a lower reproductive success than either parental species. In these cases, selection should favor individuals who reliably discriminate between the two populations and mate only with their own type. Put another way,

selection will favor the evolution of additional reproductive barriers, minimizing the probability of hybrid mating still further. This process, where natural selection acts to increase the amount of reproductive isolation between populations, is called reinforcement (<u>Servedio and Noor 2003</u>). Once two populations have begun to diverge, reinforcement can complete the process of speciation.

To find evidence of geographic isolation, scientists have to unearth clues about how geographical barriers arose in the distant past. Among the best understood of these barriers is the Isthmus of Panama. Before 15 million years ago, North America and South America were isolated continents, and the Pacific and Atlantic were joined by a seaway. Marine animals in the region could move back and forth between the two oceans. Starting 15 million years ago, the isthmus gradually rose above the ocean; and about 3 million years ago, the two oceans were cut off from each other entirely.

Nancy Knowlton and her colleagues study the animals that live today in the waters on either side of Panama. They worked out the phylogeny of some shrimp species and found that many species were more closely related to species on the other side of Panama than they were to species in their own ocean (FIGURE 10.7). You'd expect this pattern from geographic isolation, because species were split into eastern and western populations by the Isthmus of Panama.



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A: Before about 3 million years ago, the Atlantic and Pacific Oceans were joined by a seaway. Some species of shrimp had ranges that extended into both oceans. When the Isthmus of Panama formed, it isolated Atlantic and Pacific populations of some of these species of shrimp. B: Genetic studies show that the closest relatives of some shrimp species in the Atlantic are not other Atlantic shrimp, but Pacific species. These studies are evidence of allopatric speciation: the formation of new species by geographic barriers. (Information from Knowlton et al. 1993.)

But were these separated shrimp populations distinct species? For allopatry to lead to speciation (at least according to the biological species

concept), reproductive barriers need to accumulate in the geographically separated species, so that they wouldn't interbreed even if they were brought back together. To test the mating behavior of the shrimp, Knowlton brought some of these Pacific and Atlantic species pairs to her laboratory. When she placed them in tanks together, they did not interbreed (Knowlton et al. 1993).

Typically, speciation takes thousands or millions of years to split a species fully in two. But it's possible to glimpse a little of the process in experiments. In the 1970s, for example, Larry Hurd and Robert Eisenberg of Cornell University ran an experiment with a swarm of *Drosophila* flies. They put a thousand flies into a chamber that had traps at its top and bottom. As they buzzed around in the chamber, some of the flies accidentally ended up in the top trap and some in the bottom one. Hurd and Eisenberg waited three hours, until 50 insects had flown into each trap. They then removed the traps from the chamber and allowed the flies in the traps to mate—but only with other flies that had flown in the same direction.

Hurd and Eisenberg reared the new generation of flies and released it back into the chamber. Again they collected 50 flies from each trap and bred them, then repeated the experiment for the next generation of each set. They put the offspring of the upward-flying flies in the chamber and collected the first 50 flies to breed a new generation; they did the same with the downward-flying ones. For 16 generations, they repeated this process. The results were striking. The tendency for the flies to go up or down became much stronger. While it took three hours to collect the first batch of 50 flies, it took only 10 minutes after 16 generations.

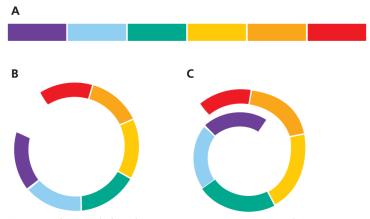
Now Hurd and Eisenberg wondered how distinct the two populations had become. To find out, they mixed the up and down populations together—creating a lab version of sympatry, as it were. Nearly all of the flies chose to mate with flies that had traveled in the same direction. A preference had evolved, in other words, that would reduce the flow of genes between the two populations (Hurd and Eisenburg 1975).

Rings of Species

Allopatric speciation—the splitting of populations through geographic isolation—is a major factor in the origin of species. But under some conditions, reproductive barriers can evolve even when the ranges of populations are not completely divided. One way this can occur is if the populations of a species stretch so far that the ones at opposite ends start to diverge.

The populations of a species at the northern end of its range may face bitter winters while the populations at the southern end bask in blazing summers. Interbreeding will carry some alleles from the north to the south. But alleles that promote survival in the cold won't be favored by natural selection in the south the way they are in the north. As long as the northern and southern populations are evolving away from each other faster than gene flow across the extent of the range can homogenize them, the populations will continue to diverge.

Evolutionary biologists have long wondered what would happen if a species' range expanded so that the two ends came into contact. The populations in those two ends might be so different from each other—either through selection or drift—that they couldn't interbreed. Researchers dubbed this case a "ring species." (FIGURE 10.8).

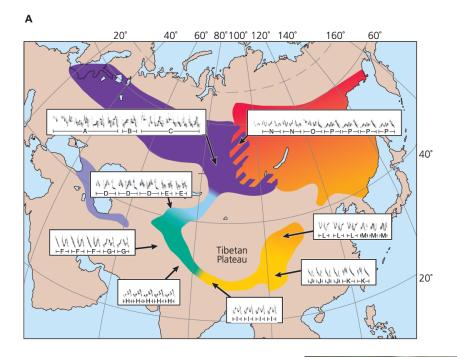


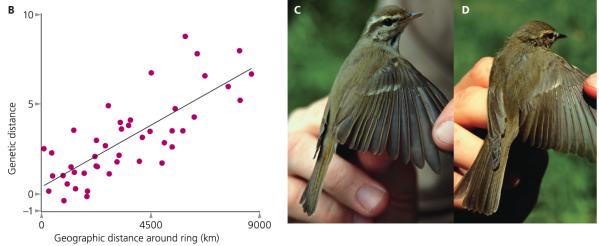
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Ring species can evolve from a connected series of populations (A), each of which can interbreed with its neighboring populations. Once they diverged sufficiently from each other, the populations at both ends of the range can no longer interbreed. If the two ends of the range wrap around and overlap, they create a "ring." Although they live side by side, the individuals from the two populations at the ends remain reproductively isolated (C).

Ring species are not just theoretical possibilities. Darren Irwin, a biologist at the University of British Columbia, and his colleagues have been traveling to Russia, Mongolia, China, and Nepal to document an actual ring species formed by populations of a small bird known as the greenish warbler (*Phylloscopus trochiloides*). They have spent years observing the bird's colors, listening to its songs, and sampling its DNA.

In analyzing the warbler genes, Irwin discovered that the oldest populations are found in the southern end of their range, along the south face of the Himalayas. There, in one of the few regions in Asia where suitable forests still grew, the birds probably survived during the last ice age. As the glaciers retreated about 20,000 years ago and forests expanded northward, the greenish warblers expanded as well. Some birds expanded around the east side of the Himalayas, up through China, into eastern Siberia. The other branch moved around the west side, into Central Asia. The Tibetan Plateau to the north of the Himalayas has remained dry and treeless, and so the greenish warblers did not colonize that region. Instead, they spread like two arms of a circle until the arms overlapped at last in central Siberia (FIGURE 10.9).





Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: (C) and (D) Jessica H. Irwin.

Greenish warblers originally evolved south of the Himalayas (A). They later spread eastward and westward along the mountain range and then expanded their range north. Today, ornithologists recognize six subspecies of greenish warblers (shown here in different colors). The subspecies have diverged in male song pattern (represented here in boxes) and female preferences for those patterns along the two paths of their expansion. Populations are also more genetically distant the farther they are from each other around the ring (B). In Siberia, the two arms of the species range overlap. But the

western Siberian warblers (C) and the eastern ones (D) rarely interbreed. That's because they sing different courtship songs and have different patterns on their feathers. They behave like two separate species, and they are as genetically distant from each other as they would be if they were at opposite ends of their range. They are, in other words, a striking example of a ring species. (Data from <u>Irwin et al. 2005</u>.)

Irwin recorded the songs of greenish warblers across the bird's range. He found differences in the songs from one population to the next. The song diverged independently in each arm of the bird's range. As a result, where the two ends of the greenish warbler's range overlap, the sympatric birds sing markedly different songs. If an east Siberian male warbler hears another east Siberian male singing, it responds aggressively. But if it hears a west Siberian male, it makes no response. Females apparently prefer the songs of their own males, judging from the fact that the east and west Siberian warblers almost never interbreed. Despite living in the same place, they produce almost no hybrids.

Irwin has also collected blood from greenish warblers in order to analyze markers in their DNA. As he moved along each arm of the bird's range, he found new markers, demonstrating that the greenish warblers had diverged across their range. At the two ends of the arms, the birds are most genetically distant. Even though birds from the two endpoints now intermingle, they remain genetically distinct. That's what you'd expect if the greenish warbler were a ring species. The two ends would not interbreed, despite their overlapping range (Irwin et al. 2005).

Ring species preserve in their rings living relics of the transitional sequence of speciation. They display side by side the stages by which a common ancestral population diverged into reproductively isolated descendant populations. In essence, ring species portray across space a process of speciation that occurs through time, providing rare glimpses of the mechanism of speciation.

Speciation in Sympatry

In the 1960s, as evidence for allopatric speciation was starting to pile up, Ernst Mayr argued that allopatry was almost always essential for new species to emerge. But some scientists disagreed. They suggested that populations could diverge without any geographic separation at all, in a process called sympatric speciation.

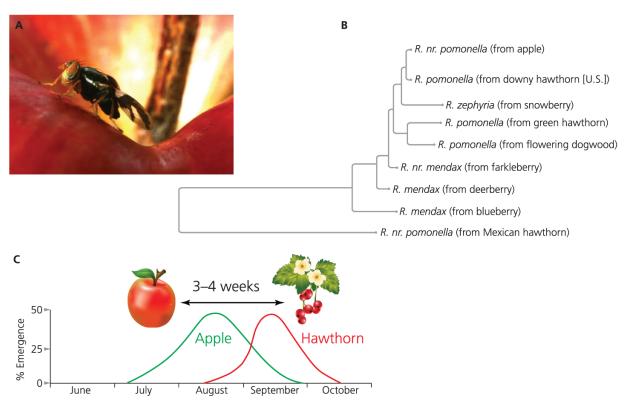
The debate over allopatric and sympatric speciation has been roiling ever since. In a few instances, researchers have made a compelling case that actual populations have diverged without geographic separation. The low number of these reports doesn't necessarily mean that sympatric speciation is rare; it may simply be hard to document. Making matters even more complex, it appears that some species have evolved through a combination of allopatric and sympatric speciation. For example, they may have diverged initially in allopatry and did not complete their isolation due to reinforcement until after they later became sympatric. In these cases, researchers might end up studying contemporary populations that live in sympatry without realizing that the initial divergence between them actually had occurred while they were geographically separated. Today, biologists generally agree that speciation in sympatry is possible, based on theoretical models of population genetics, although it's more complex than speciation in allopatry.

One of the best-documented cases of sympatric speciation is a genus of flies, known as *Rhagoletis*, that lives in North America. *Rhagoletis* flies rendezvous around fruit trees to find a mate. The females lay their eggs on fruit hanging from the trees. After the fruit falls to the ground and starts to rot, the eggs hatch, and the larvae feed on it. The insects then crawl into the soil to pupate. They go into a kind of suspended animation known as diapause to survive the winter and then emerge as adults in the spring to find another fruit tree.

In the northeastern United States, *Rhagoletis* forms two sympatric—but genetically distinct—populations (known as host races). One host race lives only on hawthorn trees; the other lives on apple trees, where it has earned the name "apple maggot fly." The two host races are specialized in many ways

for their particular fruit. Apples and hawthorns blossom at different times of year, and the host races come out of diapause at different times as well. The flies live in the same fields and travel past both plants as they search for their preferred host—a host they recognize by the odor, color, and once they land, size of the fruit. Thus they are considered truly sympatric.

Apples are new to North America. English colonists brought apple trees with them from the Old World less than 400 years ago. In the mid-1800s, farmers in the Hudson River Valley in New York reported *Rhagoletis* pomonella flies infesting their apple trees. Over the next 50 years, the apple race of *Rhagoletis* spread through the eastern United States, becoming a serious agricultural pest (FIGURE 10.10).



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FIGURE 10.10

A: One population of *Rhagoletis pomonella* flies in the eastern United States lays its eggs exclusively on hawthorn fruit. Another lays eggs only on apples. Given that apple trees were introduced to the region only 400 years ago, the apple-laying flies must have evolved from a native hawthorn population. B: A phylogeny of the flies shows how the

apple specialists are related to *R. pomonella* populations that lay their eggs on other trees. C: Apple and hawthorn trees bloom at different times of year. In each population of flies, selection favors flies that develop so as to coincide with the host fruit. This selection helps isolate the populations, allowing them to diverge in sympatry.

To understand how the new race of flies evolved, Jeff Feder, of the University of Notre Dame, and his colleagues have studied the variation in the hawthorn flies. *Rhagoletis* lives on 19 different species of hawthorn trees across a vast range, from Canada down to Mexico. In different regions, the flies are genetically programmed to emerge from diapause at different times and to recognize different hawthorn fruit odors. As a result, the hawthorn flies already had preexisting genetic variation that made a shift to apples possible. Flies that happened to emerge from diapause when apples were beginning to grow sometimes landed on them instead of hawthorns. They could now feast on a fruit with less competition from other *Rhagoletis* flies (Feder and Filchak 1999). They also could avoid attacks from parasitoid wasps that specifically search for hawthorns in order to find *Rhagoletis* larvae in which they can lay their eggs.

Feder and his colleagues argue that natural selection favored alleles that could allow the flies to take more advantage of the apples, shifting their diapause even further away from that of hawthorn flies. Apple-preferring flies evolved to favor odors that are emitted by apples, but not by hawthorn (Linn et al. 2003). As a result, they showed a strong fidelity to the apples, returning to the fruit to lay their eggs (Feder et al. 1994). Their preferences for different fruit created an isolating barrier to gene flow, even though both races lived in the same geographic area.

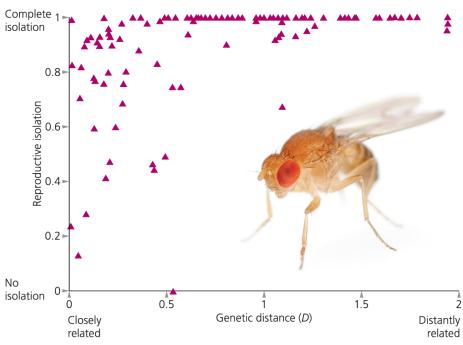
Ecology plays a major role in Feder's hypothesis. The *Rhagoletis* populations are diverging not solely through the accumulation of mutations through genetic drift; they are adapting to different ecological niches, and that adaptation is giving rise to reproductive barriers. Other species of *Rhagoletis* show signs of being more advanced in this process of ecological speciation. They attack other plants, such as flowering dogwoods, blueberries, and snow-berries, showing even stronger differences in diapause and their preferences for fruit odors. These reproductive barriers also have led them to become more genetically distinct. After only four

centuries, the apple maggot fly and hawthorn maggot fly are considered separate races of *Rhagoletis pomonella*. But with enough time, Feder argues, they could become separate species as well.

The Speed of Speciation

The evolution of reproductive barriers of the sort we've described so far is a gradual process. When a barrier first evolves, it may be very weak, only slightly reducing gene flow. Eventually, other barriers evolve, and over time they become stronger, gradually choking off gene flow.

To estimate how long it takes reproductive barriers to lead to new species, Jerry Coyne, a population geneticist at the University of Chicago, and H. Allen Orr, a population geneticist at the University of Rochester, studied *Drosophila melanogaster* and related species of flies. They reviewed studies in which scientists had put males and females of different species together and observed whether they successfully reproduced. All told, they tallied the performance of 171 species pairs, giving them a score of 0 to 1. Species that never had hybrid matings scored a 1. If species mated freely with each other, they scored 0. Most pairs fell somewhere in between. Coyne and Orr then used the molecular clock (see Chapter 7) to calculate how long ago each pair of species had descended from a common ancestor. FIGURE
10.11 shows their results. They found it can take as long as several hundred thousand years for two populations of flies to become isolated enough to be considered true species by the biological species concept (Coyne and Orr
1997).



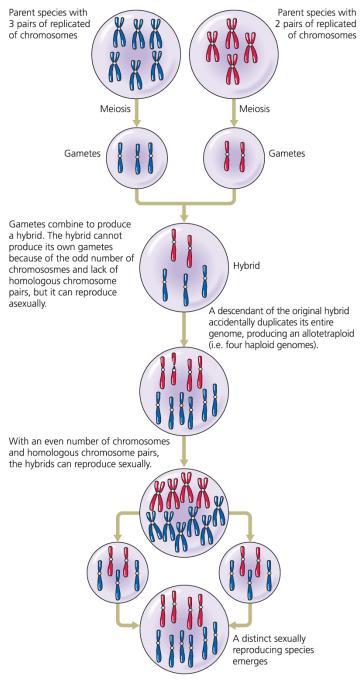
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This graph shows how reproductive isolation evolved among species of *Drosophila*. The genetic distance (*D*) between two species increases with time. It takes roughly a million years for *D* to reach a value of one. By then, a typical pair of *Drosophila* species no longer interbreed. (Data from Coyne and Orr 2004.)

Speciation sometimes takes hundreds of thousands of years, but in other cases it can take just a few generations. Many species of plants have a biology that enables them to speciate virtually instantly. If a pollen grain lands on a flower of a different species, it may succeed in fertilizing an ovule, producing a hybrid offspring. In many cases, the hybrid is sterile. In other cases, the hybrid can breed with one of its parental species. But in still other cases, the hybrid can evolve into a new species.

FIGURE 10.12 shows how this can happen. A species with two pairs of chromosomes mates with a species with three. The hybrid cannot produce viable gametes, because it cannot evenly divide its five chromosomes. But in some cases, hybrids can reproduce asexually. The hybrid's offspring accidentally duplicate their entire genome, resulting in 10 chromosomes.

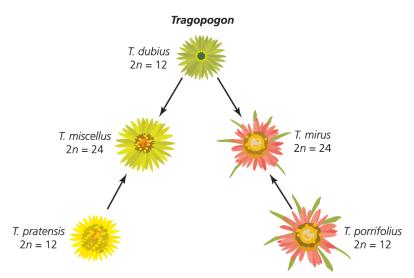
Now the chromosomes can pair and produce viable gametes. Those gametes can come together to produce new offspring with 10 chromosomes that can reproduce sexually. But the offspring can't interbreed easily with the two species that produced it. In other words, it has become a distinct, sexually reproducing species.



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Alloploidy is a common form of speciation that occurs when two species hybridize. If the resulting hybrid offspring have an odd number of chromosomes, they cannot reproduce sexually. But they can still reproduce asexually. If a mutation causes the DNA in the asexual hybrid to be duplicated, it can reproduce sexually.

This process is called allopolyploidy. Botanists sometimes call allopolyploidy "instant speciation," although studies on plants indicate that the process can be more complex and drawn out (Soltis and Soltis 2009). In some cases, two species produce many such hybrids, which then interbreed among themselves. The multiple independent origins of the hybrids give the new allopolyploid species added genetic variation, which allows natural selection to work more effectively. While there's much left to learn about how allopolyploidy produces new species, one thing is clear: it's surprisingly common. Perhaps half of the estimated 300,000 flowering plant species evolved through allopolyploidy (FIGURE 10.13).



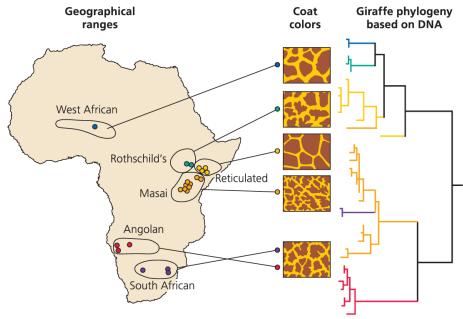
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FIGURE 10.13

Salsify flowers (*Tragopogon*) have experienced speciation by allopolyploidy a number of times. Here are two examples of how the flowers have hybridized to produce new species (*T. miscellus* and *T. mirus*). (Information from <u>Soltis and Soltis 2009</u>.)

Uncovering Hidden Species

As scientists learn more about speciation, they use this knowledge to do a better job of classifying the world's biological diversity. Rick Brenneman and his colleagues are studying the diversity of giraffes by collecting several different lines of evidence about the animals. One of these lines is giraffe DNA. They collected tissue from 266 giraffes across Africa, from which they sequenced 1707 nucleotides from the DNA of each animal. From the giraffe DNA, the scientists produced an evolutionary tree, illustrated in **FIGURE**10.14. Their results indicated that the giraffes separated from a common ancestor about a million years ago. Its descendants diverged into six lineages with distinct variations in coat patterns.



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FIGURE 10.14

Rick Brenneman and his colleagues have reconstructed the evolution of giraffes from a common ancestor. As this map shows, giraffes have diverged into several lineages, each with a distinct range and coat color. The scientists propose that they are six different species. (Information from <u>Brown et al. 2007</u>.)

Brenneman and his colleagues argue that these results indicate that the giraffes form six separate species, according to the phylogenetic species concept (Figure 10.14). To verify that the giraffes are reproductively isolated, which is the main criterion of the biological species concept, they looked for hybrids. The West African, Angolan, and South African lineages are too geographically distant ever to interbreed with the other giraffes. But the three lineages in East Africa live close to each other and could, at least theoretically, produce hybrids. Brenneman and his colleagues found only three hybrid giraffes—less than 1 percent of the animals they darted. For the most part, the lineages of giraffes had not mixed. Brenneman and his colleagues speculate that the different coat patterns help to keep the lineages from interbreeding because giraffes are choosing to mate only with other giraffes having the same pattern (Brown et al. 2007).

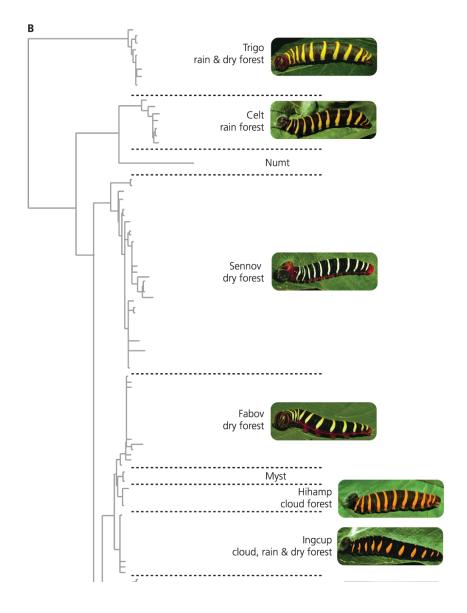
If the scientists are right about the number of species, conservation biologists trying to save giraffes from extinction face a much bigger problem than they previously realized. Rather than trying to preserve a single species spread across most of Africa, they may need to come up with strategies tailored for each of the six different species. Rothschild's giraffe, for example, which lives in Uganda and Kenya, has dwindled down to a few hundred individuals. Importing giraffes from other regions won't save this species; the others will simply replace it.

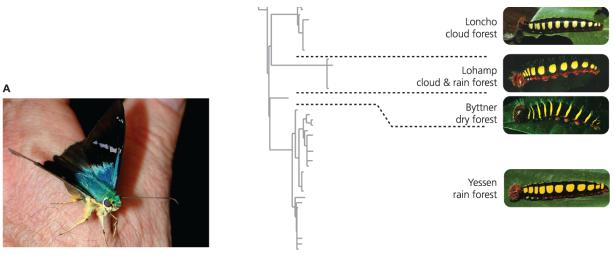
This unexpected diversity in giraffes is not a fluke. Molecular biology is enabling scientists to discover many other species hiding in plain sight. In Costa Rica, for example, Daniel Janzen and his colleagues at the University of Pennsylvania have been trying to document the true extent of the diversity of insects. In one study, they examined the neotropical skipper butterfly, *Astraptes fulgerator*. This handsome blue and black insect was first described as a species in 1755, and it has been recorded living as far north as the United States and as far south as Argentina.

Over 25 years, Janzen has reared thousands of skipper caterpillars, and he noticed that they feed on a wide range of plants. Caterpillars are usually very fussy about which plants they feed on, because they produce chemicals that are finely tuned for overcoming defensive chemicals in plants. It seemed odd that the neotropical skipper butterfly would be able to feed on so many

species. A closer look revealed that caterpillars that prefer the same plants tend to have the same color patterns. And Janzen and his colleagues found subtle differences among the adult forms that the caterpillars developed into. Janzen hypothesized that the single species of *A. fulgerator* might be six or more species.

To test whether these so-called cryptic species exist, Janzen joined forces with Paul Hebert, a biologist at the University of Guelph in Canada who identifies species by looking for distinctive stretches of DNA. In 2004 they reported that *A. fulgerator* formed 10 distinct genetic clusters, each of which produced a distinct color pattern as caterpillars (Hebert et al. 2004). Janzen argues that these clusters are, in fact, separate species (FIGURE 10.15).





Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photos by Dan Janzen. "Ten species in one: DNA barcoding reveals cryptic species in the neotropical skipper butterfly Astraptes fulgerator" by Herbert et al. PNAS, 2004:Vol. 101, No. 41, pp.14812–14817.

A: Skipper butterflies in Costa Rica produce bright colors as adults. B: An analysis of their DNA reveals that the butterfly populations have diverged from one another, revealing a diversity of species that had previously gone unnoticed. (Information from Hebert et al.2004.)

Many of the caterpillars that Janzen has captured over the years have turned out to be infected with parasitoid wasps. Adult female wasps inject the eggs into caterpillar hosts, where they hatch and develop as larvae, feeding on the still-living caterpillars. Janzen wondered if the wasps might form cryptic species as well. Janzen, Hebert, and their colleague James Whitfield, from the University of Illinois, analyzed 2597 different wasps (Smith et al. 2008). Upon inspecting the wasps visually, the scientists recognized six different genera of wasps parasitizing the caterpillars. On closer examination, they were able to sort them into 171 provisional species. But DNA barcoding revealed 142 more species, bringing the total to 313. What had once been considered a single species—a tiny black wasp known as *Apanteles leucostigmus*—turned out to be 32 species.

While results like this are exciting, Janzen and other scientists also know that they can't happen soon enough. Biodiversity, especially in tropical rain forests, is facing extraordinary threats from humans, such as logging operations and growing cities (see <u>Chapter 11</u> for more on extinctions).

Janzen and his colleagues want to know how many species there are in places like Costa Rica, so that they can figure out how to save as many of them as possible.

The Puzzle of Microbial "Species"

Over the past century, evolutionary biologists studying species and speciation have focused most of their attention on animals and plants. The concepts they've developed for these taxa have proven difficult to apply to other parts of the tree of life. The biological species concept is based on male and female individuals interbreeding. But males and females don't exist among bacteria and archaea. Instead, they mainly reproduce asexually, producing identical or nearly identical clones.

Traditionally, microbiologists have defined species based on morphological and biochemical traits. A species of bacteria might be distinguished by a round shape, an ability to grow on a particular sugar, and its dependence on oxygen. Today, however, those methods have been supplanted by DNA. Microbiologists have used the phylogenetic species concept to identify microbial species. They draw evolutionary trees and identify species at the tips of the branches.

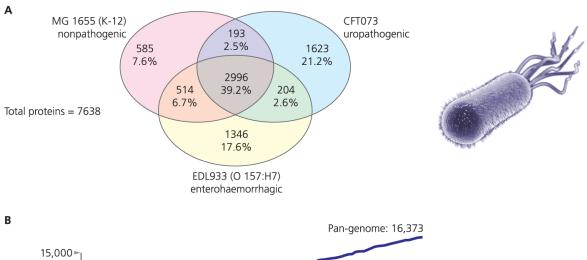
Like so much about the study of species, this method is not perfect. Imagine a flask of *E. coli* sitting in a lab. A mutation arises as a microbe divides. Now there's a lineage in the flask that can be distinguished from all the others. It wouldn't make sense to declare that lineage a new species. To avoid this absurd situation, microbiologists have come up with other standards. One widespread method involves examining a gene for a ribosomal RNA molecule called 16s rRNA. If a microbe's 16s rRNA gene is 97 percent or more identical to a known species, then it is assigned to that species. If it is less than 97 percent identical to any previously discovered 16s rRNA sequence, on the other hand, the microbe is considered a new species.

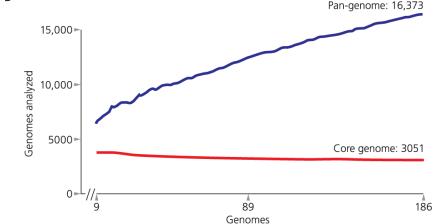
By this standard, the diversity of microbes is vast and mostly unknown to science. A single spoonful of soil may have 10,000 different species of bacteria—more species of bacteria than have been formally named and described (Oren 2004). Scientists cannot yet estimate how many species of prokaryotes there are worldwide. Each survey they carry out—whether it is in a lake, in the Arctic tundra, or in the human body—continues to yield many

DNA sequences that do not closely match the sequence of anything found before. Scientists have also recognized that many microbial species are rare and therefore may be missed by surveys that aren't intensive enough (Sogin et al. 2006). There are likely millions—perhaps even hundreds of millions—of microbial species on Earth.

Yet the 97 percent cutoff has no biological significance. It's just an arbitrary standard. If microbial "species" are real units, they'll need a more solid foundation. And there's an even more profound challenge to any species concept for microbes: horizontal gene transfer.

Microbiologists first discovered horizontal gene transfer in the 1940s, but at first it seemed like a limited phenomenon involving relatively few genes. When scientists developed methods for sequencing entire genomes, it became abundantly clear this was not the case. In 2002, for example, Fred Blattner of the University of Wisconsin and his colleagues published a paper comparing three *E. coli* genomes (Welch et al. 2002). One was a harmless lab strain called K12. The second, O157:H7, can cause bloody diarrhea and organ failure and is spread in contaminated food. The third, called CFT073, causes urinary infections. Blattner and his colleagues found many genes in each strain that had no homologs in either of the others. Out of 7638 genes present in at least one of the strains, only 2996 (39.2 percent) were present in all three strains (FIGURE 10.16). This pattern is the result of different strains acquiring genes through horizontal gene transfer, while also losing other genes through accidental deletions.





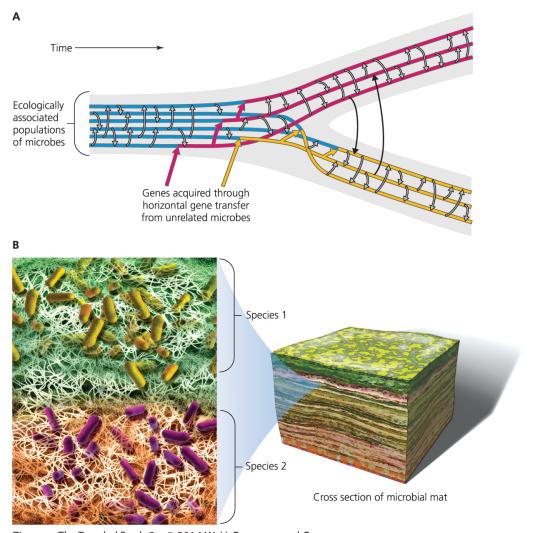
Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: luismmolina/Getty Images.

A: After the genomes of three strains of *E. coli* were sequenced in 2002, scientists discovered that they share only a limited "core" of genes. The total set of *E. coli* genes—known as the "pan-genome"—was much larger than any one strain's genome (Welch et al. 2002). B: As of 2012, scientists had sequenced 162 *E. coli* genomes. This graph shows that the pan-genome has grown rapidly as scientists have added more genomes to their analysis, while the core genome has shrunk. The pan-genome will probably continue to grow in years to come as scientists study more strains of *E. coli*. Their research is a striking demonstration of how horizontal gene transfer transforms genomes of bacteria, even closely related forms. (Data from Kaas et al. 2012.)

As scientists add more *E. coli* genomes to their analysis, this core continues to shrink, and the total number of *E. coli* genes—the pan-genome, as researchers call it—continues to expand. In a 2012 study, David Ussery of

the Technical University of Denmark and his colleagues analyzed 168 *E. coli* strains (Kaas et al. 2012). They identified 16,373 genes in the pan-genome. The core genome has shrunk to 3051 genes. The phylogenetic species concept is useful only if organisms reliably pass down their genes to their offspring. If genes are moving frequently from one lineage to another, life seems to blur into a jumble of mosaic-like genomes (Doolittle and Zhaxybayeva 2009).

Despite this complexity, some scientists are optimistic that they can develop a biologically sound concept of species for microbes (Fraser et al. 2009). Like animal or plant species, microbe lineages adapt to specific ecological niches (FIGURE 10.17). In a hot spring, for example, one lineage may be selected for a particular combination of temperature, sunlight, and pH. Nearby, another lineage may be selected for a different combination (Cohan 2006). These lineages acquire new genes through horizontal gene transfer, but this process is not a random onslaught of DNA. Most of the transfers happen between closely related lineages. On rare occasion, transferred genes open up a new niche for a lineage. It evolves in a new direction—becoming, one could argue, a new species (Shapiro et al. 2012).



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FIGURE 10.17

Despite rampant horizontal gene transfer, some scientists argue that microbial species are real entities. A: This diagram shows how new species of microbes can evolve. An ancestral species (blue lines) occupies an ecological niche. The populations of the species experience the same selection pressures. They frequently trade genes through horizontal gene transfer (small white arrows). Selection preserves these transferred genes because they're well adapted to the species' niche. Sometimes horizontal gene transfer delivers a gene from a distantly related species (red and gold arrows). These foreign genes may enable microbes to occupy a new niche, and selection will favor further mutations that better adapt them to it. The species splits as populations adapt to different niches. (Adapted from Shapiro et al. 2012.) B: This process may account for the ecological patterns microbiologists find among microbes in the wild. This figure shows

how bacteria are distributed in a hot spring in Yellowstone National Park. Different species are adapted to particular temperatures and concentrations of different minerals and other nutrients.

Understanding speciation is crucial to understanding the natural world. It gives rise to the planet's biodiversity, which is currently facing massive threats that we'll examine in the next chapter. To understand how to preserve giraffes, butterflies, and other groups of organisms, we must first know their true diversity. Our own bodies harbor a huge amount of diversity, too. They are home to perhaps thousands of species of bacteria, archaea, protozoa, and fungi (Grice and Segre 2012). Our health depends on the services these invisible passengers provide, such as breaking down food and fighting off pathogens. We cannot yet put a precise figure to the number of species we harbor, because it still so difficult to say what a microbial species is. If we can resolve this ambiguity, we may be able to better understand these friends —as well as our enemies, the pathogens that make us sick (Chapter 15). And finally, understanding the nature of species can help us better understand ourselves. It's easy to see that *Homo sapiens* is a distinct species from our closest relatives, the great apes such as chimpanzees and gorillas. But as we'll see in Chapter 14, that clarity exists because we share such a distant common ancestor with apes, one that lived perhaps 7 million years ago. When we turn to our closer relatives—extinct humans such as Neanderthals —things get fascinatingly messy.

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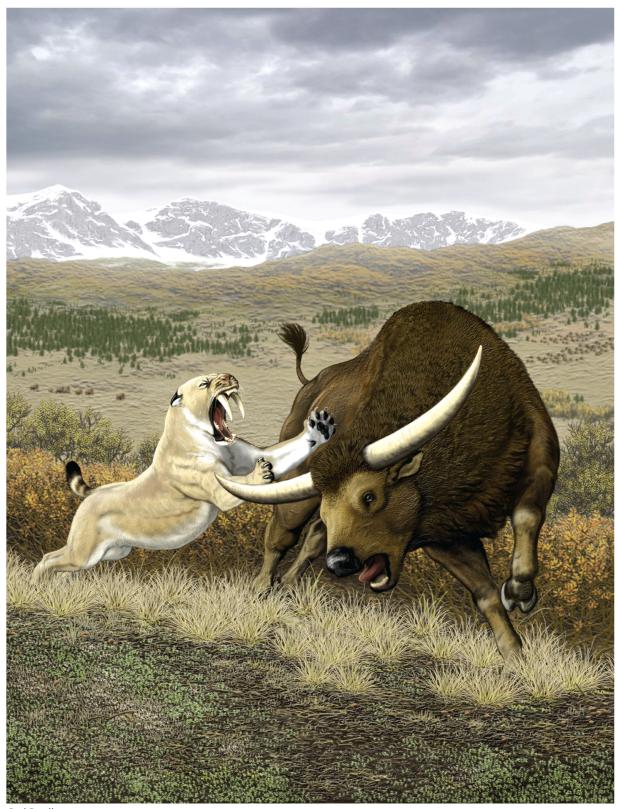
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Macroevolution

11

Life Over the Long Run



Carl Buell.

As recently as 12,000 years ago, North America was home to a wide range of large mammals such as this saber-toothed cat (*Smilodon fatalis*) and giant bison (*Bison latifrons*). These and many other species of large North American mammals have become extinct, probably due at least in part to the arrival of human hunters on the continent.

Anthony Barnosky crawls into caves and climbs mountains in search of fossils. The fossils he's after are the mammals that lived in North America during the Pleistocene, the period that lasted from 2.6 million years ago to 11,700 years ago. Barnosky, who teaches at the University of California, Berkeley, and his colleagues have found a great many mammal bones over the years. Their fossil collection includes species ranging from tiny rodents to giant mastodons. And yet for Barnosky, the fossils he *doesn't* find are just as interesting as the ones he does.



Jessica Blois.

FIGURE 11.1

Anthony Barnosky of the University of California–Berkeley explores caves with his colleagues to reconstruct the changing biodiversity of North America over the past 30 million years.

For millions of years, North America was home to a menagerie of mammals that would seem more at home on the African savanna than the North America we are familiar with today. Along with the mastodons and other elephant relatives, there were saber-toothed cats, camels, and rhinoceroses. They left behind fossils spanning vast stretches of time typically a million years or more. But there comes a point in the fossil record where each of those species simply disappears. In some cases, Barnosky and his colleagues can see evidence of a new species branching off from a vanished species before it became extinct. And sometimes—such as at the end of the Pleistocene—many species became extinct at once, without leaving any descendants. Barnosky is charting the pace of those extinctions. From their timing, he hopes to better understand what drove all those remarkable animals to oblivion.

Today there are 8.7 million species on Earth, according to a recent estimate, and many scientists suspect the final figure is far higher (Mora et al. 2011). Yet the biodiversity that we see around us today is only a tiny remnant of the vast number of species that have existed since life first emerged over 3.5 billion years ago. Evolutionary biologists estimate that over 99 percent of the species that ever existed on Earth are now extinct. Over those 3.5 billion years, the diversity of life has undergone many changes. It has risen and fallen, sometimes slowly, sometimes abruptly. Major new lineages have emerged and spread across the globe, only later to shrink into obscurity. We are left today with no end of fascinating questions. Why are almost all marsupial mammals found only in Australia and New Guinea? Why are there so many beetles (300,000 species at minimum) in the world, but so few ticks (just 878)?

The history of biodiversity—the processes and patterns of originations, adaptations, and extinctions—is known as

macroevolution. In this chapter, we'll look at some important lessons that have emerged from the study of macroevolution—not just for understanding the deep history of biodiversity but also for understanding its future. The research of scientists such as Barnosky has revealed that we humans are driving species extinct at a worrying rate, a rate that could potentially increase to truly catastrophic levels in the years to come.

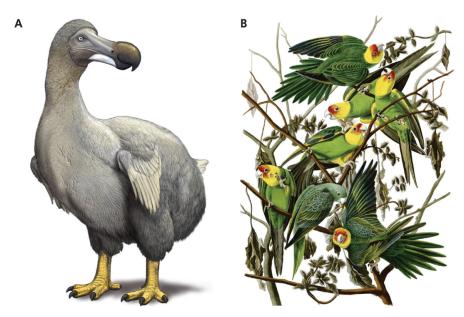
Biodiversity: The Balance between Origination and Extinction

New species are gradually emerging all around us (<u>Chapter 10</u>). Populations are becoming reproductively isolated and adapting to different niches. Whether any particular population becomes a new species over the course of thousands or millions of years depends on the specific details of its natural history and its environment. But with millions of species on Earth, evolution can steadily add new species to the world's biodiversity, like a faucet dripping water into a bucket.

Biodiversity's bucket also has leaks. As new species are emerging, old ones are gradually disappearing. The most striking proof of extinction is in the fossil record. We do not have to worry about being attacked by *Tyrannosaurus rex*, for example, because it vanished 65 million years ago. Evolutionary biologists can gain clues to how species became extinct in the past by studying living species. A species is a lineage made up of linked populations. It can endure for millions of years even though the total number of individuals in the species may fluctuate wildly over time—booming when a new source of food becomes available, or shrinking under attack from a particularly nasty parasite. Even if one population completely disappears, there are other populations to sustain the species and expand its range. If the total number of individuals in a species shrinks too far, however, it faces the risk of disappearing altogether.

Once a species falls below this threshold, any number of different factors may drive it extinct. If a lizard species is made up of just 50 individuals living on a single tiny island, a big hurricane can kill them all in one fell swoop. Small populations also face threats from their own genes. As we saw in Chapter 6, they can become vulnerable to genetic drift, which can fix harmful mutations, lowering the populations' average reproductive fitness. Small populations also have less genetic variation, which can leave them less prepared to adapt quickly to a changing environment (Allendorf et al.2012).

Scientists have documented all these processes in living species, and they've even managed to document some actual extinctions. When Dutch explorers arrived on the island of Mauritius in the 1600s, for example, they discovered a big, flightless bird there called the dodo (FIGURE 11.2). They killed dodos for food or sport and also drove down their numbers by introducing rats to Mauritius, which ate the dodos' eggs. As adult and young dodos alike were killed, the population shrank until only a single dodo was left. When it died, the species was gone forever (Rijsdijk et al. 2009).



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FIGURE 11.2

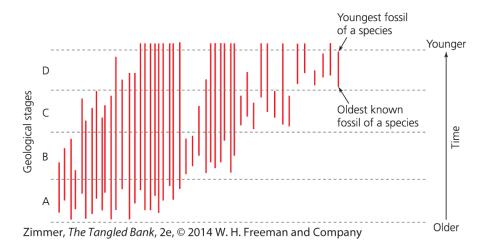
Species have become extinct throughout the history of life, including in just the past few centuries. A: The dodo became extinct in the late 1600s, probably due to hunting and the introduction of rats. B: The Carolina parakeet became extinct in the early 1900s, due in part to logging, which removed the hollow logs where it built its nests.

Simply killing off individuals is not the only way to drive a species toward extinction. Habitat loss—the destruction of a particular kind of environment where a species thrives—can also put a species at risk. The Carolina parakeet once lived in huge numbers in the southeastern United States. Loggers probably hastened its demise in the early 1900s by cutting

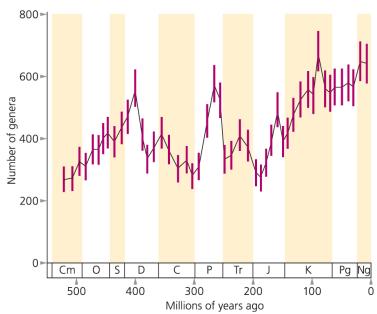
down the old-growth forests where the parakeets made their nests in hollow logs. And as we saw in <u>Chapter 1</u>, the river dolphin became extinct in China in recent years due to a combination of causes including overfishing, pollution, and habitat loss.

Scientists have carefully documented only a modest number of extinctions, because extinctions are so hard to study. It can take decades or even centuries for species to disappear, and it's rare for scientists to be able to follow a species so carefully for such a long period of time. Making matters worse, scientists so far have formally described only about 2 million species. Thus the vast majority of species have yet to be discovered. If they become extinct before then, we'll never know they ever existed.

The fossil record, likewise, provides us with only a sampling of the world's past biodiversity. But scientists have developed statistical tools to use those fossils to estimate how many species there were at different points in the past. They can also estimate the rate at which new species originate and then become extinct (FIGURE 11.3). In 2008, for example, an international team of scientists analyzed records of 3.5 million fossils of marine invertebrates over the past 540 million years (Alroy et al. 2008). They tallied how many genera were present during each geological interval. Among marine invertebrates, at least, biodiversity is higher today than it was half a billion years ago. But their increase was not steady. Their diversity sometimes jumped, sometimes slowed, and even crashed from time to time (FIGURE 11.4).



This hypothetical diagram shows how species are preserved in the fossil record. By looking at the earliest and oldest fossils of species, paleontologists can estimate not only how long species endure, but the rate at which new species evolve and old ones become extinct.



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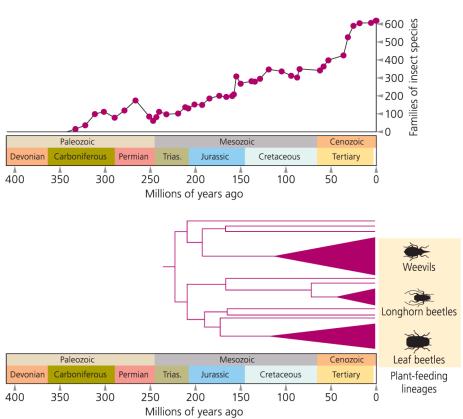
FIGURE 11.4

A team of paleontologists analyzed 3.5 million fossils of marine invertebrates to estimate the history of their biodiversity. As this graph shows, diversity has risen and fallen several times, but today there are about twice as many genera as there were at the beginning of this period. The bars are margins of error. (Data from <u>Alroy et al. 2008</u>)

Many different processes can cause these changes in diversity. If the estimated number of species goes up over the course of 10 million years, for example, the cause may be an increase in the rate at which new species originate. Think of our biodiversity bucket: you can raise the water level by increasing the flow from the faucet. But let's not forget the holes in the bucket —namely, the species becoming extinct. If the extinction rate drops, it's as if someone plugged some of the holes. The water can rise in the bucket even if the flow from the faucet doesn't change.

Even when the fossil record shows no change in diversity, there are different possible explanations. It's possible that only a few new species are emerging, balanced by an equally low rate of extinctions. It's also possible that massive extinctions are occurring, while new species are evolving at a sky-high rate. Later in this chapter, we'll look in detail at some of the jumps and falls in <u>Figure 11.4</u> and consider the explanations scientists have developed for them.

To create the graph in Figure 11.4, scientists examined the biodiversity of a wide range of clades. Focusing on a single clade also reveals intriguing patterns. FIGURE 11.5 shows a diagram plotting the diversity of insects since the earliest signs of their existence 400 million years ago. The rise of insect diversity is all the more striking when you compare them to their closest relatives, a group of arthropods called entognathans that includes springtails. The entognathan lineage is just as old as the insect lineage. And while there are a million known insect species, there are only 10,600 entognathan species.



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Insects are the most diverse group of animals on Earth. A: Insect diversity has grown gradually over the past 400 million years. B: Plant feeding may have helped spur insect diversity, because many insect lineages that evolved the ability to eat plants became more diverse than their closest relatives. This tree shows the relative diversity of plant-feeding insects and their closest relatives. (Data from Mayhew 2008.)

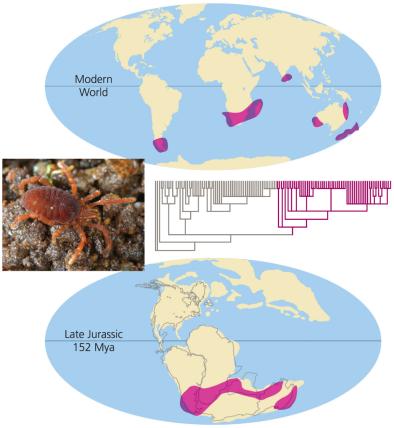
A number of biologists have probed the history of insects to determine what factors account for their huge diversity. Peter Mayhew, a biologist at the University of York, has tested various hypotheses. Insects don't seem to have a particularly high rate of speciation, he has found, but they do seem good at withstanding extinctions (Mayhew, Jenkins, and Benton 2008). Fifty percent of all families of insect species alive today existed 250 million years ago. None of the families of tetrapod species alive 250 million years ago exists today; all have been replaced by newer groups.

So what gives insects their sticking power? Mayhew argues that a few key factors are at work. The ability to eat plants provides insects with a huge amount of food; plant eating has evolved several times among insects, and the plant-eating lineages tend to accumulate more species than closely related lineages of insects that don't eat plants. The small bodies of insects may lower the amount of food they need to survive and shorten the time they need to develop from eggs. Wings also allow insects to disperse much farther than arthropods that can only crawl or jump. Mayhew argues that all these advantages gave insects a massive edge, allowing them to colonize new habitats quickly and survive catastrophes.

Riding the Continents

Few people have heard of the mite harvestman, and fewer still would recognize it at close range. It is related to the far more familiar daddy longlegs, but its legs are stubby rather than long, and its body is about as big as a sesame seed. On the floors of the humid forests where it dwells, it looks like a speck of dirt. As unglamorous as the mite harvestman may seem, however, it has a spectacular history to unfold.

An individual mite harvestman may spend its entire life in a few square meters of forest floor. The range of an entire species may be less than 100 kilometers (60 miles) across. Yet there are 5000 species of mite harvestman, and they can be found on five continents and a number of islands. Sarah Boyer, a biologist at Macalester University in Minnesota, and her colleagues have traveled around the world to catch mite harvestmen, and they've used the DNA of the animals to draw an evolutionary tree. Their results seem bizarre. One lineage of mite harvestmen, for example, is found only in Chile, South Africa, and Sri Lanka—countries separated by thousands of kilometers of ocean (FIGURE 11.6).



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One lineage of mite harvestmen can be found on continents and islands separated by thousands of miles of ocean. They reached their present locations thanks to continental drift. Around 150 million years ago, the ranges of these invertebrates formed a continuous belt. Later, the continents broke apart and moved away, taking the mite harvestmen with them. The pink branches represent lineages that are found in the regions marked in pink on the maps. (Information from <u>Boyer et al. 2007</u>.)

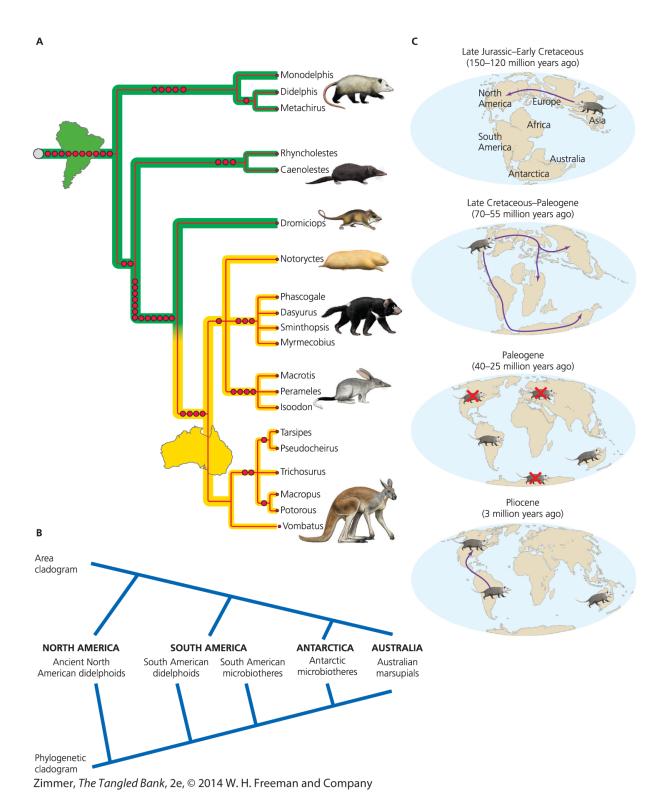
To make sense of Boyer's results, you have to think back in time. The continents have been moving slowly across the planet since they first formed, sometimes forming supercontinents and sometimes breaking into fragments. Hundreds of millions of years ago, Chile, South Africa, and Sri Lanka were joined together. The mite harvestman lineage that Boyer studies first originated there and then drifted apart along with the landmasses on which the animals lived (Boyer et al. 2007).

The study of how biodiversity has spread around the world is known as biogeography. Mite harvestmen illustrate one of the most common patterns in biogeography, called vicariance: populations become separated from each other when geographical barriers emerge. In the case of mite harvestmen, the barriers are expanding oceans. Mountains can also divide species, as well as spreading deserts and shifting rivers. The other major pattern in biogeography occurs when species themselves spread away from their place of origin. This process is known as dispersal. Birds can fly from one island to another, for example, and insects can float on driftwood.

While dispersal or vicariance alone may explain the distribution of some species, the distribution for most species is the result of a combination of the two processes. Take the case of marsupials. Although most living species of marsupials are found today in Australia, the oldest marsupial fossils come from China and North America. Today in all of North America there is only one species of marsupial (the Virginia opossum). China has no marsupials at all. How did we get to this puzzling situation?

We can understand this pattern by combining several separate lines of evidence: from studies on the molecular phylogeny of living marsupials, from the fossil record, and from the movement of the continents.

In 2010, Maria Nilsson of the University of Munster and her colleagues published a detailed molecular phylogeny of all the major marsupial groups in the world (Nilsson et al. 2010). To produce their phylogeny (FIGURE 11.7A), they examined marsupial DNA. In particular, they looked at mobile elements, those segments of DNA with the capacity to make copies of themselves that can be reinserted in other parts of the genome. They found some mobile elements that were shared uniquely by some species of marsupials, but not by others. What's more, the pattern they uncovered was a nested hierarchy, in which the mobile elements revealed clades within clades. These results were compelling evidence that the mobile elements accurately reflect the phylogeny of marsupials. What's more, this phylogeny offers clues to the movement of marsupials. All Australian marsupials form a single clade, nested within the clade of South American marsupials. This pattern is consistent with a single dispersal of Australian marsupials from South America (Figure 11.7).



Marsupials evolved through a mix of vicariance (the movement of continents) and dispersal (spreading from one landmass to another). A: Molecular phylogeny shows that

Australian marsupials form a clade nested within South American marsupials. The only living North American marsupial, the Virginia opossum (*Didelphis*), is also nested within the South American clade. (Dots denote genetic elements called retroposons that are uniquely shared by various marsupial clades. Adapted from Nilsson et al. 2010.) B: An area cladogram shows the supercontinent Gondwana broke up into today's continents. A phylogeny based on fossils matches the geological pattern. C: By combining evidence, we can construct a scenario for the evolution of marsupials.

Other scientists have studied fossils to reconstruct the phylogeny of marsupials. They've been able to incorporate extinct species that have left no DNA behind for scientists like Nilsson to study. Their studies support the hypothesis that marsupials moved from South America to Australia. They also provide more detail about the exact route. Paleontologists have found marsupial fossils from Antarctica, dating back to a time when the continent was warm enough for forests to grow. These Antarctic marsupials turn out to be more closely related to Australian species than South American ones.

The fossil record also shows that extinct North American marsupials belong to the deepest of the branches. This branching pattern parallels the order in which these continents separated from each other. North America split off first; next, South America separated from Australia and Antarctica; and last, Australia and Antarctica became separate landmasses.

These separate lines of evidence all support the same scenario for the evolution of marsupials (Springer et al. 2011). Marsupial-like mammals were living in China by 150 million years ago, the age of the oldest fossils yet found. By 120 million years ago, they had dispersed into North America, which at the time was linked to Asia. Many new lineages of marsupials evolved in North America over the next 55 million years.

From North America, some marsupials dispersed to Europe, even reaching as far as North Africa and Central Asia. All of these Northern Hemisphere marsupials eventually died out in a series of extinctions between 30 and 25 million years ago (Beck et al. 2008). Another group of North American marsupials dispersed to South America around 70 million years ago.

From South America, this branch of marsupials dispersed into Antarctica and Australia, both of which were attached to South America at the time. Marsupials arrived in Australia no later than 55 million years ago, the age of

the oldest marsupial fossils found there. Later, South America, Antarctica, and Australia began to drift apart, each carrying with it a population of marsupials (vicariance). The fossil record shows that marsupials were still in Antarctica 40 million years ago. But as the continent moved nearer to the South Pole and became cold, these animals became extinct.

Meanwhile, marsupials in South America diversified into a wide range of forms. Their ranks included a number of large species, including a strikingly catlike sabertooth—a case of convergent evolution (page 207). Those marsupial sabertooths became extinct, along with many other unique South American marsupials. The fossil record indicates that their extinction came after North and South America reconnected a few million years ago. Placental mammals expanded down through the Isthmus of Panama and established themselves in South America. It's possible that they outcompeted similar marsupial species, driving them extinct.

But that doesn't mean that marsupials were somehow intrinsically "inferior" to placental mammals. Many species of small and medium-sized marsupials still live in South America today, and one South American marsupial, the Virginia opossum, even migrated back into North America, where it now thrives.

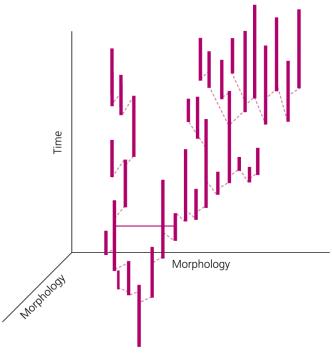
Australia, meanwhile, drifted in isolation for over 40 million years. The fossil record of Australia is too patchy for paleontologists to say whether there were any placental mammals in Australia during that time. Abundant Australian fossils date back to about 25 million years ago, when all of the mammals in Australia were marsupials. It was not until 15 million years ago that Australia moved close enough to Asia to allow placental mammals—rats and bats—to begin colonizing the continent. These invaders diversified into many ecological niches, but there's no evidence that they displaced a single marsupial species that was already there.

The Pace of Evolution

As marsupials spread out over the planet, they diversified into a range of forms, from tree kangaroos to the catlike sabertooths. Did these changes occur at a steady rate, or did the evolution of marsupials speed up and slow down along the way? Until the early 1970s, most paleontologists would have replied that macroevolution unfolded steadily. But in 1972, two young paleontologists at the American Museum of Natural History named Niles Eldredge and Stephen Jay Gould offered a new idea: perhaps macroevolution occurs in fits and starts (Eldredge and Gould 1972).

Eldredge and Gould pointed out that while the fossil record showed lots of change *between* species, it lacked much evidence of long-term, directional change *within* a species. Eldredge illustrated this point with a careful study he carried out on trilobites. One of the traits used to distinguish one species of trilobite from another in the fossil record is the number of columns in their compound eyes and the pattern those columns take. He counted the rows of columns in fossils from a single species of trilobite species and found that the number did not change over 6 million years.

Eldredge and Gould proposed that this pattern in the fossil record reflected real stasis during evolution. They weren't arguing against natural selection, of course. But they did argue that natural selection rarely pushed species out of stasis. Instead, it allowed populations to adapt to their local conditions. When small populations became isolated and branched off as new species, however, they acquired small but measurable changes in their morphology (FIGURE 11.8).



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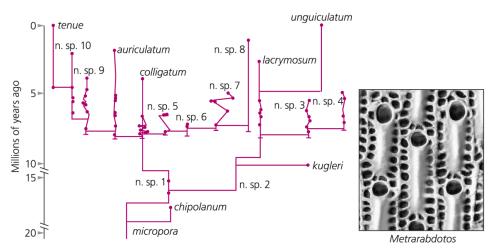
In 1972 Niles Eldredge and Stephen Jay Gould published an influential paper in which they argued that macroevolution was dominated by a pattern of punctuated equilibria. Species experienced long periods of stasis, punctuated by rapid morphological change during speciation. This is in contrast to the traditional model of slow, steady directional change in fossil lineages. (Information from <u>Eldredge and Gould 1972</u>.)

The two scientists dubbed this phenomenon punctuated equilibria. They argued that it could explain the patterns found in the fossil record—not just the long stretches of stasis, but also the appearance of new species. If morphological change occurred in just a few thousand years in small populations, the odds were low that these transitional forms would leave fossils behind.

Eldredge and Gould inspired later paleontologists to put their hypothesis to the test. It's easier said than done, however, for two reasons. One is that such a test demands exceptionally good fossil records. If paleontologists find a fossil from a lineage of oysters every 5 million years, they can know little about all the changes that happened during the intervals. Even when

paleontologists did find fossil records that were dense enough, they then had to devise a statistical measure of evolutionary change both within species and between them.

In 1986 Alan Cheetham, a paleontologist at the Smithsonian Institution, published a study that's widely considered the closest thing to an ideal test case for punctuated equilibria (Cheetham 1986). He studied the evolution of bryozoans, small animals that grow in crustlike colonies on submerged rocks and reefs. The genus *Metrarabdotos* has left a dense fossil record over the past 20 million years in the Caribbean. It's even possible to identify pairs of ancestor and descendant species living at the same time. Cheetham took measurements of several different traits on the fossils, using statistical methods to give each fossil a single number to represent its similarity to the other fossils. FIGURE 11.9 shows Cheetham's results. Just as Eldredge and Gould had predicted, he found little change within species. Most of the change must have happened as new species evolved.



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: Information from Tempo of Evolution in a Neogene Bryozoan: Rates of Morphologic Change Within and Across Species Boundaries Alan H. Cheetham Paleobiology Vol. 12, No. 2 (Spring, 1986), pp. 190–202.

FIGURE 11.9

Paleontologists have found some examples from the fossil record that can be explained by a pattern of punctuated equilibria. This diagram was produced from a study on animals called bryozoans (specifically, the genus *Metrarabdotos*). The location of the dots along the x-axis reflects their body shape; closely spaced dots indicate fossils with similar morphology. The y-axis shows the age of the fossils and their lineages. The fossil

record indicates that bryozoan species experienced little directional, long-term change over millions of years, punctuated by relatively rapid speciation in a matter of thousands of years. (Information from <u>Cheetham 1986</u>.)

Unfortunately, cases like this one are scarce, making it impossible at this point to say whether punctuated equilibria are the dominant pattern in evolution or are found in only a few lineages. In the opinion of many experts, no one mode of speciation dominates the history of life (<u>Erwin and Anstey</u> 1995).

Nevertheless, Eldredge and Gould remain hugely influential. They inspired many paleontologists to explore stasis and change in other parts of the fossil record. Gene Hunt, also of the Smithsonian, analyzed studies on 53 evolutionary lineages ranging from mollusks to fishes to primates. In each study the scientists measured a trait, such as the width of a tooth, over millions of years. A large, steady shift in the trait would be evidence for directional selection. Very little change would represent stasis. If neither directional selection nor stasis were present, the trait would fluctuate without any clear trend.

Hunt concluded that only 5 percent of the fossil sequences showed signs of directional change (FIGURE 11.10). The other 95 percent was about evenly split between random walks and stasis. The Hunt study supports the idea that stasis is a major feature in the history of life (Hunt 2007).

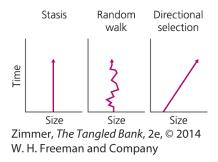


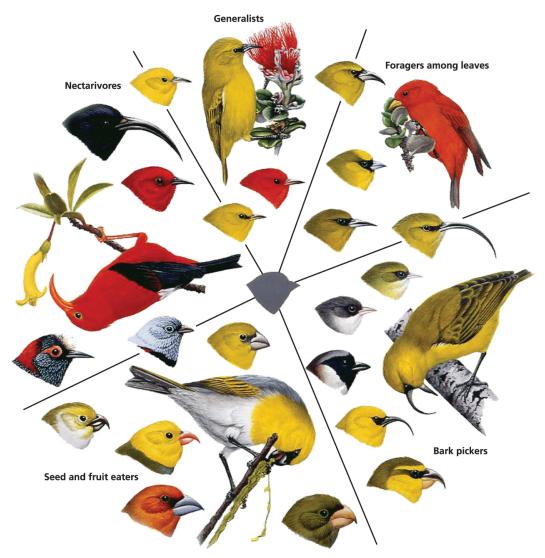
FIGURE 11.10

A trait, such as size, may experience stasis, or undergo small changes that don't add up to a significant shift, or experience long-term selection in one direction. One large-scale survey of the fossil record found that directional selection could explain only 5 percent of the data.

Adaptive Radiations

The kind of morphological changes that Eldredge and Gould sought to explain with punctuated equilibria were not terribly spectacular. A new species of trilobite emerges with a slightly different shaped eye. The holes on the surface of bryozoans grow slightly wider. But the history of life also has been punctuated by dramatic bursts of evolution. During these bursts, known as adaptive radiations, a clade diversifies into many lineages, each taking advantage of a different ecological niche (Losos 2010).

The clearest examples of adaptive radiations have taken place when a new habitat emerges. When islands rise from the sea, for example, they are bare patches of earth without an ecosystem. They are then colonized by microbes, fungi, plants, and animals from other landmasses. The arriving species diversify as they occupy the growing number of ecological niches the new island has to offer. Hawaii, for example, is home to plants and animals that underwent bursts of evolution. **FIGURE 11.11** shows one such adaptive radiation, the honey-creeper birds, which diversified into more than 50 dramatically different forms found today only on the Hawaiian Islands.



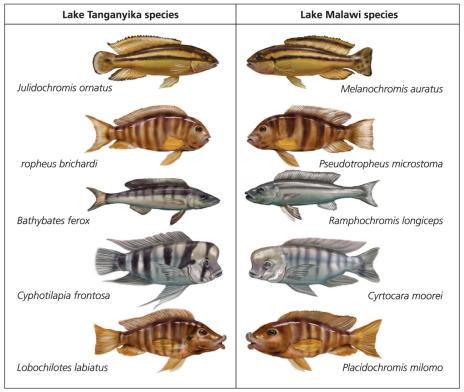
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Volcanic islands often foster adaptive radiations. Ancestral finches colonized the Hawaiian archipelago roughly 5 million years ago and diversified into more than 50 species of honeycreepers with diverse colors, feeding habits, and bill forms. More than half of these species have since gone extinct. (Information from Losos and Ricklefs 2009.)

Lakes are like aquatic versions of islands. As the climate and geology of a region changes, dry land can become submerged, opening up ecological opportunities for species that can colonize it. The Great Lakes of East Africa,

for example, were formed only over the last few hundred thousand years. After the lakes formed, cichlid fish moved into them from nearby rivers. Those colonists exploded into hundreds of species found nowhere else. Some crush mollusks, some suck crabs out of crevices, some scrap algae from rocks, and some chew the scales off other cichlids (<u>Salzburger et al.</u> 2005).

What's especially intriguing about adaptive radiations is that they often repeat themselves. The cichlids that invaded Lake Malawi were not the same ones that invaded Lake Victoria. Yet their descendants evolved into many of the same forms. Their convergence suggests that the similar ecology of the lakes controls the outcome of the adaptive radiations in them (FIGURE 11.12).



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FIGURE 11.12

Adaptive radiations sometimes lead to impressive examples of convergent evolution, as separate lineages fill up the same ecological niches. Over the past few hundreds of thousand years, cichlid fishes independently colonized the Great Lakes of East Africa and evolved striking similar forms.

Scientists can study the adaptive radiations in lakes and on islands in great detail because they've occurred recently, and their boundaries are clear. The fossil record also offers evidence of possible adaptive radiations in the distant past taking place across much larger ecosystems. When a new ecological opportunity has emerged, lineages have sometimes responded with a burst of new species that display an unprecedented range of variations. When grasslands first emerged around 20 million years ago, for example, horses underwent an adaptive radiation. They evolved into large and small sizes, and their teeth took on different shapes that they could use to feed on different kinds of vegetation in the new ecosystem (MacFadden and Hurlburt 1988).

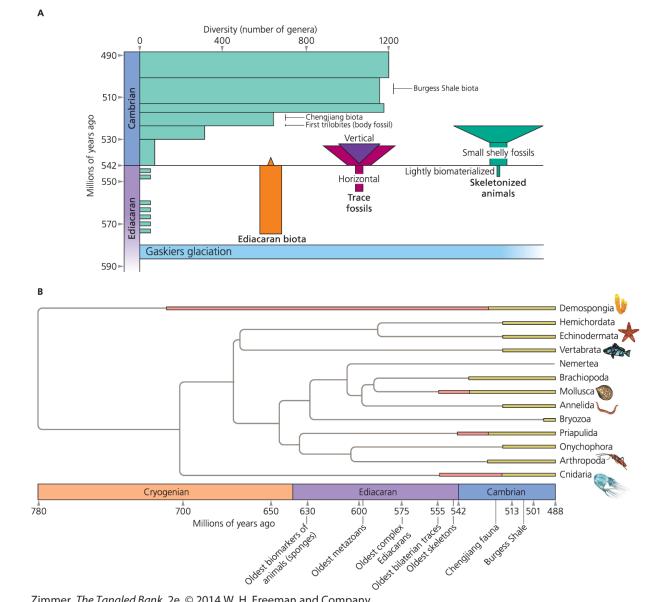
Sometimes the occupants of ecological niches have to disappear before a lineage can experience an adaptive radiation. That appears to be what happened 65 million years ago, when the large dinosaurs and marine reptiles became extinct (a subject we'll revisit later in this chapter). Once those animals were gone, the opportunity arose for other animals to take on their former roles—as large herbivores, for example, or top predators in the ocean. Mammals got that chance. Before the end of the Cretaceous, no mammal had been bigger than a raccoon. After the Cretaceous, elephant-sized herbivores evolved, along with large carnivores (Smith et al. 2010). Mammals even returned to the oceans for the first time, evolving into whales and seals that preyed on fish.

The Cambrian Explosion

The diversification of mammals since the end of the Cretaceous has unquestionably been spectacular, and yet, in the scheme of things, it was fairly modest. Every species in that adaptive radiation—be it rhinoceros, manatee, or flying fox—remained a mammal with the same hallmark features such as hair and milk. But 540 million years ago, a far more dramatic diversification took place—one that gave rise to many of the major groups of animals.

Scientists sometimes call the episode that took place between 543 and 510 million years ago the Cambrian Explosion. That name is a bit of a misnomer, because it conjures an image of animals suddenly popping into existence. As we saw in <u>Chapter 3</u>, the fossil record for animals now extends to a time far earlier. The oldest signs of animals now date back more than 635 million years, over 100 million years before the start of the Cambrian.

Scientists are also extending the history of animal evolution with the aid of the molecular clock, which we introduced in Chapter 7. In 2011, Doug Erwin of the Smithsonian Institution and his colleagues published a large-scale analysis based on 100 species from all major living clades of animals. A condensed version of their tree is illustrated in FIGURE 11.13. The common ancestor of all animals, they estimate, lived about 800 million years ago. The first major split in animal evolution was the divergence of sponges and all other animals. The ancestors of cnidarians and bilaterians diverged about 700 million years ago. The major lineages of living bilaterians diverged from each other between about 670 and 600 million years ago (Erwin et al.2011). The molecular clock, in other words, promises that there are older animal fossils paleontologists have yet to discover.

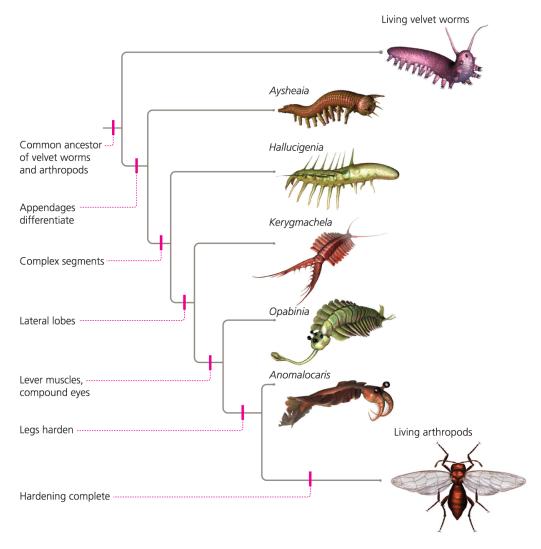


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The early evolution of animals represents one of most striking evolutionary diversifications in the history of life. A: Fossils document the growing diversity of animals. The earliest macroscopic animal fossils were left by the Ediacaran biota. In late Cambrian rocks, paleontologists find traces left by animals crawling across the seafloor and, later, burrowing down into the sediment. Small bits of skeletons also begin to appear in the early Cambrian. A burst of fossil diversity emerges around 525 million years ago. (Adapted from Marshall 2006.) B: This phylogeny is based on studies on animal DNA. The age of the nodes was determined by using the molecular clock. Tan bars show the known

fossil record of different groups. Pink bars show the range of fossils that have been proposed to belong to some groups. This research indicates that the explosion of diversity seen in the animal fossil record during the Cambrian was preceded by 150 million years of evolution that paleontologists have yet to document in much detail. (Information from Erwin et al. 2011.)

Scientists are also discovering the step-by-step evolution of the major groups of animals by studying Cambrian fossils. Arthropods, for example, are the most species-rich group of animals on Earth today: they include insects, arachnids such as spiders and scorpions, and crustaceans such as shrimp and crab. They have a combination of traits, such as a hardened exoskeleton, not seen in any other group of animals. But that doesn't mean those traits suddenly emerged all at once in the Cambrian. Early relatives of arthropods had only some of their traits, but not others. In FIGURE 11.14, we can see how these fossils help document the evolution of the arthropod body plan.



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The fossil record documents how major groups of animals emerged during the Cambrian. Arthropods—a group that includes insects, spiders, and crustaceans—share a number of traits, such as jointed exoskeletons. Some Cambrian fossils were relatives of today's arthropods, lacking some of these traits. They help reveal the stepwise process by which a major group of animals evolved. (Information from Budd 2008.)

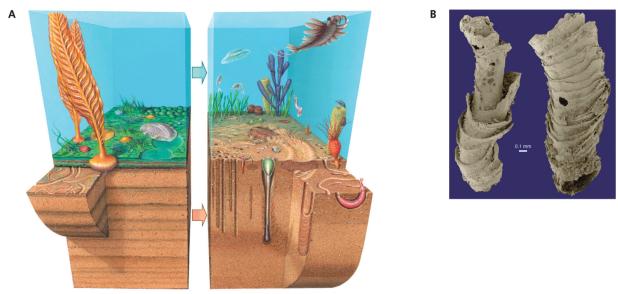
Clearly, then, the evidence overwhelmingly shows that early animals evolved incrementally over perhaps 200 million years. Still, the Cambrian was a time of extraordinary evolution in the animal kingdom. When paleontologists look at 530-million-year-old rocks, they mainly find small,

shell-like fossils. When they look at rocks just 20 million years younger, they find fossils that are recognizable relatives of living arthropods, vertebrates, and many other major groups of animals. A period of 20 million years is far longer than anything we humans can imagine. But over the course of the 3.5-billion-year history of life, it's a relatively short period. Scientists are investigating a number of hypotheses that may explain what happened. It appears that three kinds of factors worked together to produce the Cambrian Explosion: changing physical conditions, a versatile genetic toolkit, and a new kind of ecosystem (Erwin and Valentine 2013).

Being an animal requires a lot of energy. For one thing, animals need to burn fuel to make collagen, a protein that binds cells together in their bodies. And if animals are going to move around in the ocean, they will need even more energy to power their muscles. Up until about 635 million years ago, oxygen was probably too scarce in the ocean to support such an energy-demanding way of life. But geologists have found that after that point, oxygen levels rose dramatically and for reasons that are still not clear (Sahoo et al. 2012).

Early in their evolution, animals evolved the genetic toolkit we explored in <u>Chapter 8</u>. This toolkit may have allowed them to take advantage of the changes in the environment. With modest modifications to their genes, animals could develop dramatically new forms, complete with new sensory organs and appendages. Some animals could now swim quickly in the open ocean. Others began burrowing into the seafloor. These new body plans led to new relationships and new ecosystems never before seen on Earth.

The earliest animals appear to have lived like sponges do today—trapping microbes or organic matter from the water as they remained anchored to the seafloor. From these sedentary ancestors, animals with guts and nervous systems evolved, and they were able to swim through the water or burrow into the muck. Eventually, some predators evolved that began to attack other animals. FIGURE 11.15B shows a 550-million-year-old fossil, *Cloudina*, bearing the oldest known wounds from the attack of a predator. Prey evolved defenses of their own, forcing the predators to evolve new attacks. Animals got locked in a feedback loop, making their ecosystem ever more complex, opening up even more niches they could adapt to.



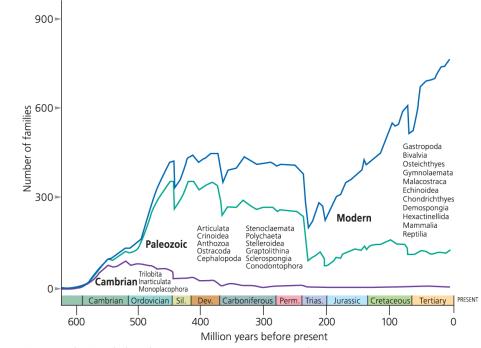
Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company "From Predatorial Borings in Late PreCambrian Mineralized Exoskeletons by Bengtson and Yue. Science, 1992: Vol. 257, pp. 367–369" Reprinted with permission from AAA S and Stefen Bengtson.

FIGURE 11.15

A: During the Cambrian, ocean ecology changed dramatically. New animals began burrowing, crawling on the ocean floor, and swimming rapidly after prey. B: The holes in these 550-million-year-old fossils of *Cloudina* were bored by predators—some of the earliest signs of predation in the fossil record.

Deciphering Life's Rises and Falls

The Cambrian period marks the start of the 540-million-year fossil record that is illustrated in Figure 11.3. Ever since the 1970s, biologists have teased clues out of that long history about why animal diversity has risen and fallen this way. In 1981, for example, Jack Sepkoski of the University of Chicago sorted the species into their major taxonomic groups. He found that the diversity of groups through time was not random. In fact, he could identify three sets of species—what he called "evolutionary faunas"—that rose and fell together with remarkable consistency (FIGURE 11.16). Sepkoski dubbed them the Cambrian, Paleozoic, and Modern faunas. These faunas were made up of different clades. The change that Sepkoski found is a bit like the change that occurred in camera technology in the early 2000s. Before then, the technology was dominated by film cameras made by Kodak, Nikon, and many other companies. But then the film "fauna" was replaced by digital cameras, also made by many different companies.



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FIGURE 11.16

Jack Sepkoski identified three "evolutionary fauna" in the fossil record since the Cambrian. The Cambrian fauna arose at the beginning of the Paleozoic and quickly declined. The Paleozoic fauna arose in the Ordovician. The so-called Modern fauna has its roots in the Cambrian but came to dominate the planet after the end of the Permian, 250 million years ago. (Data from <u>Sepkoski 1981</u>.)

The Cambrian fauna arose first. It was dominated by trilobites, inarticulate brachiopods, and coil-shelled mollusks known as monoplacophorans. Following a quick start, this fauna went into decline over tens of millions of years. By the Ordovician, Sepkoski found, the Cambrian fauna was overshadowed by the Paleozoic fauna. It included other groups of brachiopods, along with echinoderms, corals, crustaceans, ammonites, and mollusks. The Paleozoic fauna thrived until the end of the Permian, 250 million years ago.

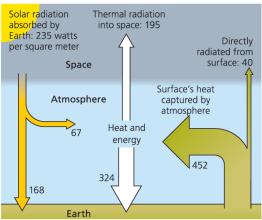
The Modern Fauna consists of clades such as gastropods (including snails), bivalves (clams), and vertebrates. It originated in the Cambrian and then later dominated the ocean.

It's intriguing to ponder why these faunas succeeded each other the way they did. One interesting clue Sepkoski found was that each fauna had a different rate of originations and extinctions. The Cambrian fauna had both high origination and extinction rates, with the result that individual species didn't last very long. The Paleozoic fauna had lower origination and extinction rates, and the Modern fauna had lower rates still. The species in today's oceans thus evolve new species slowly, but then last longer before becoming extinct. It seems, when it comes to slow and steady macroevolution, that slow and steady may win the race (Valentine 1989).

Shanan Peters, a paleontologist at the University of Wisconsin, has looked for an explanation for why the faunas succeeded each other as they did. He found that most fossils of the Paleozoic fauna are found in sedimentary rocks known as carbonates, which formed from the bodies of microscopic organisms that settled to the seafloor. Most of the Modern fauna fossils are found in rocks known as siliciclastics, which formed from the sediments carried to the ocean by rivers. Over the past 540 million years, carbonate rocks became rare, while siliciclastic rocks became common, possibly as

rivers delivered more sediment to the oceans. Peters proposes that as the seafloor changed, the Modern fauna could expand across a greater area while the Paleozoic fauna retreated to a shrinking habitat where it suffered almost complete extinction (Peters 2008).

The planet has changed in other ways that also may have played a part in shaping the history of its biodiversity. Earth's climate has warmed and cooled, for example. The climate is determined in part by the radiation delivered by the sun. But changes in the planet's orbit or its tilt can change how much radiation it receives. Once this energy reaches Earth, it can be stored as heat in the atmosphere or the ocean, or it may bounce back into space. Gases like carbon dioxide trap heat in the atmosphere and keep the planet warmer than it would be otherwise (FIGURE 11.17).



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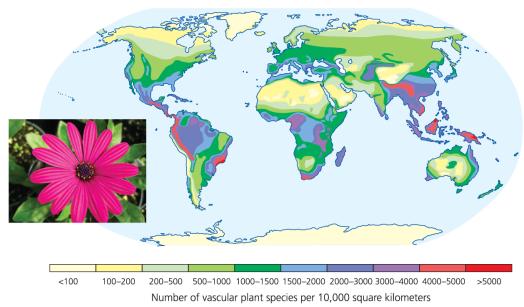
FIGURE 11.17

Earth's climate is influenced by changes in incoming radiation from the sun and the chemical composition of the atmosphere. Large changes in the climate—both warming and cooling—can drive extinctions.

Geologists can reconstruct the history of Earth's climates by looking at the chemistry of its rocks. Warm water has a higher concentration of the oxygen isotope oxygen-18 than cool water does, for example, and so rocks that form in warm water will lock in those isotopes as well. These records have demonstrated that Earth's climate has indeed fluctuated over the planet's

history. One major source of this variation is the amount of carbon dioxide in the atmosphere. When certain types of volcanoes erupt more, they deliver more of these heat-trapping greenhouse gases to the atmosphere.

Peter Mayhew of the University of York wondered if these changes in climate might affect the diversity of life. Today's biodiversity certainly hints that this might be the case. The tropics have a much higher diversity of living species of both plants and animals (FIGURE 11.18) than are found in the cooler regions close to the poles. This pattern holds on both land and sea. It's possible that the warmer temperatures foster a higher density of species. When Mayhew and his colleagues made a careful comparison of the fossil record and the climate record, they did indeed find a correlation. During warmer periods of the past 540 million years, the diversity of marine invertebrates has been higher. These researchers' analysis suggests that warmer temperature increases the origination rate over long evolutionary time scales. (Mayhew et al. 2012).



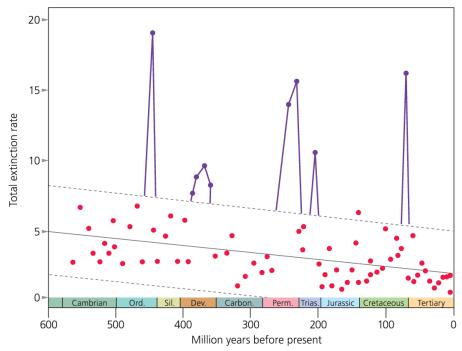
Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: Elaine Davis/Shutterstock.

FIGURE 11.18

The tropics have a higher diversity of species than other regions. This map shows the diversity of vascular plants. Some scientists argue that this pattern is partly due to the extra solar energy that the tropics receive. It might explain the association between warmer climates and more diversity in the fossil record.

The "Big Five" Mass Extinctions

Figure 11.3 displays some striking drops in diversity. The true scale of these crashes becomes clear when we look at the extinction rate over the past 540 million years (FIGURE 11.19). Although the extinction rate fluctuates, it reaches five major peaks. Vast numbers of species disappeared in these geologically short periods of time. The biggest bout of mass extinctions ever recorded, at the boundary of the Permian and Triassic 252 million years ago, is estimated to have claimed 96 percent of all species on Earth.



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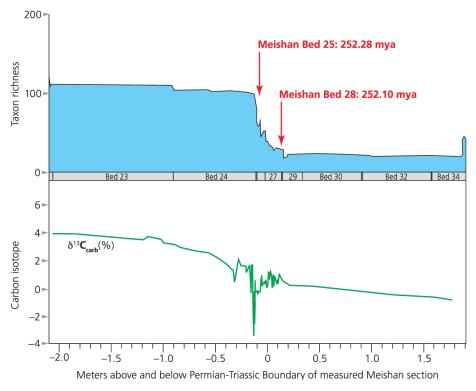
FIGURE 11.19

In 1982, David Raup and Jack Sepkoski of the University of Chicago estimated the extinction rate in marine invertebrates over the past 540 million years. In most of the intervals they studied, the extinction rate remained close to an average "background" level, shown here by dashed lines. But in five intervals, they discovered far higher extinction rates. These mass extinctions claimed over half of all species in existence at

the time. Scientists are now investigating the causes of these "Big Five." (Data from Raup and Sepkoski 1982.)

Paleontologists have long debated whether mass extinctions shared the same causes as background extinctions, or whether some fundamentally different process was responsible. To test the alternative hypotheses, they have searched for rocks that formed during those mass extinctions that may chronicle those exceptional times. The Permian-Triassic extinctions, for example, may have been driven by volcanoes. Rocks in Siberia dating back 252 million years contain huge amounts of lava that covered a region as big as the United States. They released a harsh cocktail of gases into the atmosphere that would have disrupted the climate.

Shu-zhong Shen of the Nanjing Institute of Geology and Paleontology and a team of Chinese and American colleagues discovered some of the best evidence for this disruption in Chinese rocks from the mass extinctions (Shen et al. 2011). After analyzing all the results, Shen and his colleagues concluded that the biggest mass extinctions in the history of life took place in an interval that lasted less than 200,000 years. The extinctions coincided neatly with drastic changes in the amounts of carbon dioxide in the atmosphere and oceans. As we saw in Chapter 3, geochemists can use the ratio of carbon-12 and carbon-13 isotopes in rocks to make inferences about the environment in which they formed. Shen and his colleagues found that the carbon isotope ratio changed slowly over 90,000 years before the end-Permian extinctions and then experienced an extreme shift that lasted only 20,000 years (FIGURE 11.20).



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FIGURE 11.20

The Permian-Triassic mass extinctions were the biggest known die-off in the fossil record. An estimated 96 percent of all species became extinct. *Top:* A team of Chinese and American scientists sought to determine how quickly the species disappeared. They used radiometric dating to determine the dates of 300 rocks from that age around southern China. They also measured the diversity of species at each point in time, shown here in blue. Thanks to the fine resolution of their study, they determined that the drop in diversity took less than 200,000 years. *Bottom:* To find clues to what could cause such a catastrophe, the scientists also measured the ratio of carbon isotopes. They found a drastic change, lasting only 20,000 years, at the start of the extinction period. These pieces of evidence, along with many others, suggest that carbon dioxide released by volcanoes triggered a worldwide ecological collapse. (Information from Shen et al. 2011.)

Shen and his colleagues propose that these volcanoes were responsible for the huge shift in carbon isotopes. Their eruptions released massive amounts of carbon dioxide and methane into the atmosphere. Carbon dissolving in the oceans acidified the water, disrupting the physiology of many marine organisms. At the same time, the carbon dioxide and methane in the atmosphere warmed the planet.

Mayhew has found that a warm climate correlates with high diversity, but that correlation breaks down during mass extinctions—possibly because the climate warms at such a fast rate. Instead of nurturing more diversity, the increasing heat jolts the biosphere. At the end of the Permian period, sudden warming in the oceans may have driven out much of the free oxygen in the surface waters. Some researchers have suggested that in these acidic, low-oxygen waters, once-rare types of bacteria thrived, releasing toxic gases such as hydrogen sulfide (<u>Erwin 2006</u>).

Giant volcanic eruptions are not the only things that can affect life across the planet. Mass extinctions can also come from space. In the late 1970s, the University of California geologist Walter Alvarez discovered evidence for an extraterrestrial cause, and he did so entirely by accident.

Alvarez was searching for a way to estimate precisely the ages of rocks. His father, the physicist Luis Alvarez, suggested that Walter measure levels of a rare element called iridium. Iridium falls to Earth from space at a relatively steady rate, and so it might act like a geological clock.

However, when Walter Alvarez collected rocks in Italy from the end of the Cretaceous period 66 million years ago, he discovered concentrations of iridium far higher than average. The Alvarezes and their colleagues proposed that an asteroid or comet, rich in iridium, struck the Earth at the end of the Cretaceous period (Alvarez et al. 1980). In 1991, geologists in Mexico discovered a 110-mile-wide crater along the coast of the Yucatán Peninsula of precisely that age.

What made Alvarez's discovery electrifying for many paleontologists was that the end of the Cretaceous also saw one of the biggest pulses of extinctions ever recorded. Through the Cretaceous, the Earth was home to giants. *Tyrannosaurus rex* and other carnivorous dinosaurs attacked huge prey such as *Triceratops*. Overhead, pterosaurs as big as small airplanes glided, and the oceans were dominated by whale-sized marine reptiles. By the end of the Cretaceous period, these giants were entirely gone. The pterosaurs became extinct, leaving the sky to birds, which were the only surviving dinosaurs. Marine reptiles vanished as well. Along with the giants went millions of other species, from shelled relatives of squid (called ammonites) to single-celled protozoans.

The impact on the Yucatán may have had enough energy to trigger wildfires thousands of kilometers away and to kick up tidal waves that roared across the southern coasts of North America. It may have lofted dust into the atmosphere that lingered for months, blocking out the sunlight. Some compounds from the underlying rock in the Gulf of Mexico mixed with clouds to produce acid rain, while others absorbed heat from the sun to raise temperatures.

Many researchers argue that this impact was largely responsible for the mass extinctions at the end of the Cretaceous period. But some geologists point out that not long before the impact, India began to experience tremendous volcanic activity that probably disrupted the atmosphere and the climate as well. Meanwhile, some paleontologists question how much effect either the impact or the volcanoes had on biodiversity at the end of the Cretaceous. The diversity of dinosaurs and other lineages was already beginning to drop millions of years earlier. Moreover, if a sudden environmental cataclysm wiped out the dinosaurs and millions of other species, it's strange that amphibians did not also suffer mass extinctions. Those are the animals that today are proving to be acutely vulnerable to environmental damage.

Whatever the exact causes of mass extinctions turn out to be, it is clear that they left great wakes of destruction. After the Permian-Triassic extinctions 252 million years ago, for example, forests were wiped out, and weedy, fast-growing plants called lycopsids formed vast carpets that thrived for a few million years before giving way to other plants. And when ecosystems finally recovered from the mass extinctions, they were fundamentally different than before. On land, for example, ancient reptile-like relatives of mammals were dominant before the extinctions. They took a serious blow, however, and did not recover. Instead, reptiles became more diverse and dominant—including dinosaurs, which would thrive for 200 million years.

A similar pattern unfolded 66 million years ago for the Cretaceous extinctions. After large dinosaurs became extinct, mammals came to occupy many of their niches, evolving into large carnivores and herbivores. In the oceans, mammals evolved into whales, taking the place of marine reptiles (page 6). Even as mass extinctions wipe out old biodiversity, they may open

the way for new radiations, either by wiping out predators or by clearing out ecological niches (FIGURE 11.21).

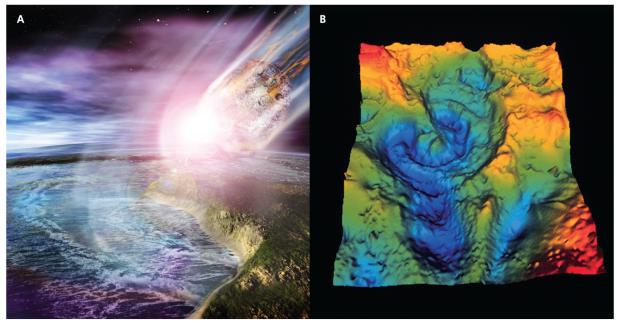


Photo: Both A and B: NASA (Chicxulab asteroid impact) Courtesy, V.L.Sharpton/LPI/NASA.

FIGURE 11.21

A: An artist's conception of what it looked like when an asteroid collided with Earth 65 million years ago. B: Among the lines of evidence for the collision are traces of the impact in rocks on the coast of Mexico.

The New Die-Off

The dodo was not alone as it headed for oblivion. Humans were also driving other species toward extinction at the same time. There are written accounts of a few hundred species that become extinct during the past few centuries, but scientists suspect that many others have also quietly vanished. Some researchers have tried to estimate the current rate of extinctions by focusing on groups of species that have enjoyed a lot of scientific scrutiny. Birds are one such group, because they're relatively big, bright, and adored by bird-watchers around the world. In 2006, Stuart Pimm of Duke University, an expert on bird extinctions, tallied the total number of bird extinctions known to have been caused by humans (Pimm et al. 2006). He looked at historical records of birds such as the dodo, but he also included extinct birds discovered by archaeologists on islands in the Pacific Ocean.

Pimm and his colleagues calculated how quickly birds were becoming extinct and compared this result to background rates of extinction documented in the fossil record. They concluded that birds are disappearing a hundred times faster. And Pimm warns that this rate will only accelerate in the coming decades. Many bird species that aren't extinct are already endangered, their populations vanishing thanks to hunting and lost habitat. Given the growing human population and the continuing deforestation in many bird habitats, Pimm fears that these endangered species will become extinct as well (FIGURE 11.22). He predicts that in a few decades, birds will become extinct a thousand times faster than the background rate.



Photo: Jamidwyer.

FIGURE 11.22

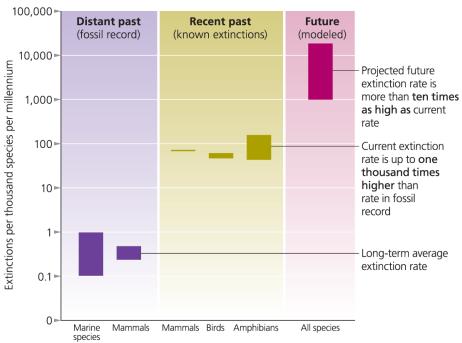
Humans have had a major effect on most of the world's habitats. Millions of acres of tropical rain forest are being cut down for timber and to clear land for farming. As the human population continues to grow, the pressure on these habitats is likely to increase.

Anthony Barnosky and his colleagues recently carried out a much wider study. In comparing the current extinction rates of all major groups of animals to the fossil record, they looked not just at background extinction rates, but mass extinctions as well (<u>Barnosky et al. 2011</u>).

Humans probably started triggering extinctions thousands of years ago. Immediately after humans arrived in North America about 15,000 years ago, for example, Barnosky and his colleagues have found that the extinction rate more than tripled. Humans likely played some part in those die-offs, helping to cause extinctions by hunting animals so fast they couldn't replace their numbers. But the rate of those extinctions was modest compared to the acceleration that has occurred over the past 500 years. The extinction rate is

now far above the background rate in the fossil record—although it's not yet at the level of the Big Five mass extinctions.

Unfortunately, hunting, pollution, and habitat destruction have driven down many species to dangerously low levels. It's possible that if those endangered species become extinct at a rapid rate, human-driven extinctions will become true mass extinctions (FIGURE 11.23). If so, we will need to change the name of the Big Five to the Big Six.



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FIGURE 11.23

The rate of extinction is now much higher than the background rate. If it increases, as many scientists now predict, we are entering a new pulse of mass extinctions. (Information from Millennium Ecosystem Assessment 2005.)

As sobering as these results may be, they may actually be an underestimate. Humans are just starting to add new pressures to the world's biodiversity that may have enormous impacts in the future. Every year, humans release more than 7 billion metric tons of carbon dioxide into the atmosphere by burning wood, oil, coal, and gasoline. Over the past two centuries, humans have raised the concentration of carbon dioxide in the air

from 280 parts per million in 1800 to over 400 parts per million in 2013. Depending on how much fossil fuel we burn in the future, levels of carbon dioxide could reach 1000 parts per million in a few decades. On a geological time scale, it's as if we've set off a gigantic carbon bomb.

All this extra carbon will have two potentially devastating effects. Some of the carbon is being absorbed from the atmosphere into the oceans (FIGURE 11.24). As a result, the pH of seawater is dropping. Bärbel Hönisch, a pale-oceanographer at Columbia University's Lamont-Doherty Earth Observatory, and his colleagues have found that the ocean is acidifying faster now than at any point in the past 300 million years (Hönisch et al. 2012). As the pH of seawater drops, the additional hydrogen ions interfere with the growth of coral reefs and shell-bearing mollusks, such as snails and clams (Zeebe et al. 2008). These animals may simply die, and the reefs may disintegrate. The collapse of coral reefs could lead to more extinctions, because a quarter of all marine animal species are sheltered by the coral reefs.

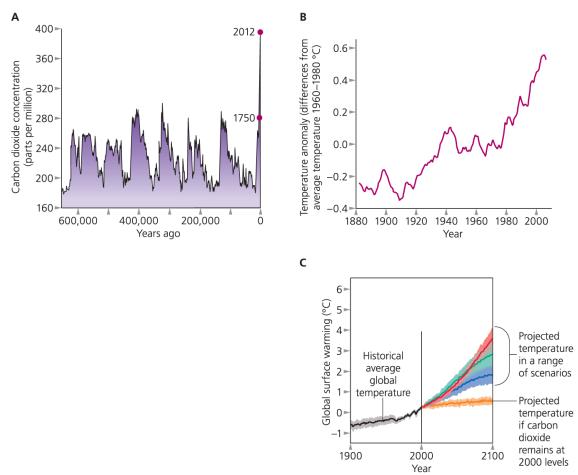


Photo: National Oceanic and Atmospheric Administration.

FIGURE 11.24

Much of the carbon dioxide humans are injecting into the atmosphere is being absorbed by the oceans, where it is lowering the pH of seawater. This change of chemistry could be devastating to coral reefs, which are home to a quarter of the ocean's biodiversity.

At the same time, carbon dioxide is warming the planet (FIGURE 11.25). Over the past century, the average global temperature has already risen 0.74°C (1.33°F). Over the next century, computer models project, the planet will warm several more degrees unless we can slow down the rise of greenhouse gases.



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FIGURE 11.25

A: Human activity has already dramatically raised the concentration of carbon dioxide in the atmosphere far beyond levels seen in the past 600,000 years. B: Thanks to the heat-trapping power of carbon dioxide, the average temperature of the planet has risen for the past century. C: Computer projections consistently show that the planet will warm much more in the next century if the concentration of atmospheric carbon dioxide continues to increase. This rapid climate change may raise the extinction rate even higher by reducing the habitat where species can find suitable temperatures and rainfall.

Animals and plants have already responded to climate change (<u>Parmesan 2006</u>). Thousands of species have shifted their ranges. Some species now live beyond their historical ranges, tracking the climate they've adapted to. Other species that live on mountainsides have shifted to higher elevations.

Scientists can't predict the effects of climate change on biodiversity precisely, but they warn that the result could be devastating. Among the first victims of climate change may be mountain-dwelling species. As they move to higher elevations, they will eventually run out of refuge. Polar bears and other animals adapted to life near the poles may also see their habitats simply melt away. In other cases, the climate envelope will shift far away from its current location. Some species may be able to shift as well, but many slow-dispersing species will not. Conservation biologists are now debating whether they should plan on moving species to preserve them (Bellard et al. 2012; Hannah 2012).

It is reasonable to ask why we should do anything to stop the coming mass extinctions. After all, extinction is a fact of life, and life on Earth has endured through big pulses of extinctions in the past, only to rebound to even higher levels of diversity. Mass extinctions are a serious matter, even on purely selfish grounds. People who depend on fish for food or income will be harmed by the collapse of coral reefs, which provide shelter for fish larvae. Bees and other insects pollinate billions of dollars of crops, and now, as introduced diseases are driving down insect populations, farmers will suffer as well—not to mention everyone else who depends on the food they produce. Biodiversity also sustains the ecosystems that support human life, whether they are wetlands that purify water or soil in which plants grow. A single species can disappear without much harm to an ecosystem, but the fossil record shows that extinctions can lead to the complete collapse of ecosystems for millions of years.

The studies by Barnosky and his colleagues show us that if we maintain our current course, we will enter the sixth great mass extinction event. But we're not there yet. In other words, we still have time to change our impact on the natural world. And we can use the insights from macroevolution to guide our actions.

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Intimate Partnerships

12

How Species Adapt to Each Other



Photo: E.D. Brodie III.

Top: Rough-skinned newts produce enough toxins to kill dozens of people. To warn off would-be predators, they bend their backs to expose their yellow bellies. Bottom: Some populations of garter snakes can attack the newts without suffering harm, thanks to their genetic resistance to the toxin. The two species are locked in a coevolutionary arms race.

Edmund Brodie Jr. first heard the story of the poisonous coffeepot in the early 1960s. His biology professor at Western Oregon University, Kenneth Walker, told him one day about three hunters found dead at their campsite in the Oregon Coast Range. There was no sign of struggle, no wounds. The only strange thing about the campsite was the coffeepot. It contained a boiled rough-skinned newt (*Taricha granulosa*).



Photo: Susan Brodie.

FIGURE 12.1

Edmund Brodie Jr. (left) and his son Edmund Brodie III study the coevolution of rough-skinned newts and common garter snakes. In this picture, they are catching newts in British Columbia with the help of Edmund III's son, Fisher.

Brodie was hungry for a research project, and so he decided to find out if the newts had killed the hunters (Brodie 2011). To catch the newts, he went to some local ponds and erected a network of metal drift fences and traps made from 5-gallon buckets. When newts encountered one of Brodie's fences,

they crawled alongside it to find a way around and then tumbled into a bucket. Brodie brought the newts back to his lab, ground their skin with a mortar and pestle, and mixed it with water. When he injected the solution into mice, he found that even at tiny concentrations the skin could kill a mouse in a matter of minutes. When Brodie then tried the skin solution on bigger animals, he found it was potent enough to kill them, too. He concluded that a single newt could easily have produced enough toxin to kill the three dead hunters.

At the time, a team of Stanford University researchers were also studying the newts, and they succeeded in identifying the compound that made the newts so deadly. Tetrodotoxin, or TTX for short, kills by locking onto receptors on neurons, thus preventing the cells from communicating with each other and leading to paralysis. Several lineages of animals, including puffer fish and poisonous snails, have independently evolved the ability to produce TTX, either as a defense against predators or as a weapon against prey.

Brodie found all of this deeply puzzling. Why should a single rough-skinned newt produce such an overwhelming amount of such a deadly poison? The first step toward an answer to that question came one day as Brodie was collecting newts from his pit traps. Sometimes he would find snakes in the buckets, much to his surprise. The snakes hadn't accidentally fallen into the buckets; when Brodie disturbed them, they were able to quickly slither away. It was as if the snakes were trying to get into the pit traps. And in one bucket, Brodie discovered why: a common garter snake was blithely eating a rough-skinned newt. Brodie caught some common garter snakes and injected them with newt skin. They could withstand a dose of TTX that would kill a far bigger animal.

Brodie moved on to other research on snakes and amphibians and eventually became a professor at Utah State University. His

son, Edmund Brodie III, grew up catching animals and eventually followed in his father's footsteps, becoming a biologist at the University of Virginia. In the 1980s, Edmund III studied the diet of snakes in the northwestern United States. He would squeeze the snakes to force up their recent meals. The main species he was studying, the northwestern garter snake (*Thamnophis ordinoides*), had a fairly dull diet of worms and slugs. But the common garter snake (*Thamnophis sirtalis*), he discovered, was loaded with a much more varied cuisine, including frogs, birds, and, most surprisingly, a rough-skinned newt.

Edmund III knew that his father had researched the toxins of rough-skinned newts, but he had forgotten that his father had found common garter snakes eating them as well. When he shared his discovery with his father, the Brodies marveled together at this strange relationship between predator and prey. Out of that conversation came a research project that has spanned the past 25 years and has revealed many astonishing details about the intertwined evolutionary history of the newt and the snake.

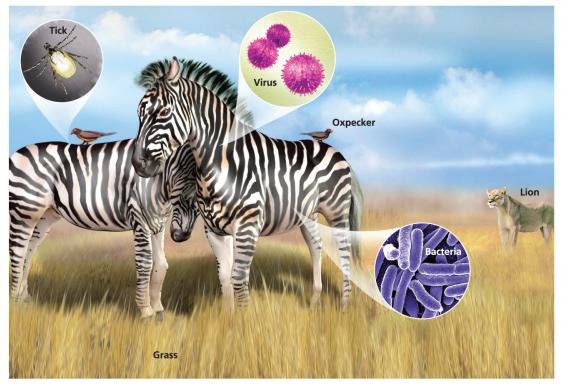
Newts and snakes are not unique in performing an evolutionary dance of this sort. Coevolution—the reciprocal evolutionary change between ecologically intimate species—has been a powerful process in the history of life. In this chapter, we look at how coevolution unfolds and examine some of the striking adaptations it has produced, such as the toxic overkill of rough-skinned newts. We'll explore the tremendous importance that coevolution has in our everyday life: viruses and other pathogens have coevolved with hosts like ourselves, while many of the foods we eat come from plants that have coevolved with insects and other animals that pollinate their flowers and spread their seeds. In fact, our own bodies are the product of coevolution: our single-celled ancestors engulfed bacteria, which have become an essential part of our biology.

The Web of Life

Every species exists in a dense web of interactions with other species. A zebra gets its energy by feeding on grasses, which in turn depend on fungi to help them extract minerals from the soil. Once the zebra chews off a mouthful of grass, the plant matter makes its way to the animal's gut, where microbes make the enzymes necessary to break it down.

Just as the grass is food for the zebra, the zebra is food for lions and other predators, along with vultures and other scavengers. If it's not killed by a predator, the zebra may die from an infection caused by deadly viruses or other pathogens. It is typically infested with ticks and other ectoparasites that suck its blood. And you'll often see oxpecker birds sitting on the zebras' backs, picking off the ticks and providing the large animals some relief.

All of these interactions have the potential to undergo natural selection. Whether they actually do depends on whether they have the necessary ingredients. As we saw in Chapter 2, natural selection requires that a trait have some effect on an organism's fitness, that it varies among individuals, and that its variation be at least partly heritable. As for fitness, all the interactions shown in FIGURE 12.2 can potentially influence the fitness of the species involved. The zebra may lower the fitness of grass by eating seeds that would otherwise have sprouted and reproduced. The oxpeckers may be able to raise the zebra's fitness by reducing the energy it would have to invest in defending against ticks. If a lion catches the zebra, its fitness will fall to zero.



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FIGURE 12.2

Each species exists in a web of ecological interactions. Each interaction can potentially raise or lower an organism's fitness and therefore can be subject to coevolution.

Ecological interactions can also vary from individual to individual. One particularly good example of this variation comes from a study that Heather Henter, then at Cornell, published in 1995 (Henter 1995). She studied wasps that lay eggs inside aphids (wasps that reproduce this way are called parasitoids). When eggs hatch, the wasp larvae feed on their still-living hosts. Just as our immune system fights off viruses, aphids fight against the larvae. Their defense consists of entombing the parasite in a suffocating ball of tissue.

Henter started her study with a single aphid, which then reproduced genetically identical clones of itself. She unleashed a group of wasps on them. The wasps laid their eggs in the aphids, and Henter waited to see how they fared. Some wasp larvae developed successfully in their hosts while others were more likely to die. In other words, they varied in how well they

coped with the same aphid genotype. The wasps Henter used in her experiment belonged to different groups of relatives, so she could compare how related wasps and unrelated ones fared against the aphids. She found that their performance was heritable: in other words, the success of a wasp tended to be similar to that of its relatives (FIGURE 12.3).



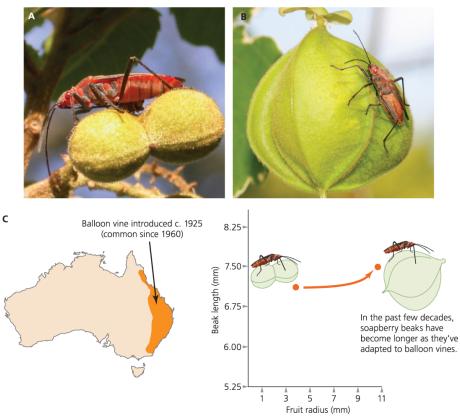
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FIGURE 12.3

The parasitoid wasp *Aphidius ervi* lays its eggs inside aphids. Aphids can sometimes kill the eggs before they hatch and start to feed on them. To study the effectiveness of the wasps, Heather Henter reared wasps from 47 different fathers. She then allowed females from each of those families to attempt to parasitize genetically identical aphids. The wasps' efforts ranged from failure to success. This graph shows the families ranked from lowest to highest parasitism. Henter's experiment demonstrates that wasps have genetic variation for success at using aphids as hosts. (Data from Henter 1995.)

Under certain conditions, scientists can actually track the evolution of one species adapting to another. In Australia, for example, native soapberry bugs drill their long beaks into native fruits to reach their seeds (Carroll et al. 2005). The shape of their beaks is precisely adapted to the fruits, which you'd expect from thousands of years of adaptation. But in the 1960s, a new plant arrived in Australia—an American species of balloon vine—that the soapberry bugs started to feed on. There was just one hitch: the balloon vine fruit is bigger than the native fruit, so the bugs had a hard time reaching the seeds.

Scott Carroll of the University of California, Davis, and his colleagues reconstructed the history of this shift in several ways. They looked in museum collections for soapberry bugs that lived in the early 1900s, before the arrival of the balloon vine. They also collected living soapberry bugs that now feed on the invasive balloon vines and compared them to insects that continue to feed only on smaller native fruit. They discovered that the insects that have shifted to the balloon vine have beaks that are 10 to 15 percent longer (FIGURE 12.4). The longer beaks are not perfectly matched to the balloon vine fruits, but the evolution that has occurred over the past few decades has raised their fitness by allowing them to get more seeds. Carroll and his colleagues offered balloon vine fruits to soapberry bugs in their laboratory. They found that longer-beaked bugs were able to feed on the seeds within the balloons at almost twice the rate of the shorter-beaked soapberry bugs that still feed on native fruits.

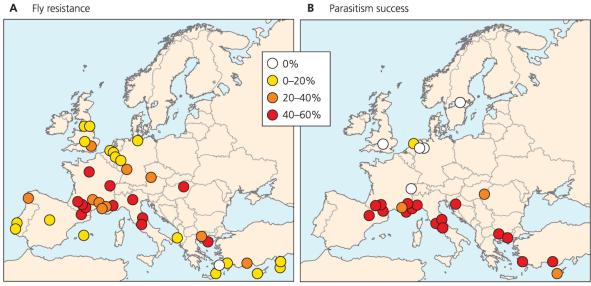


Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company A: Scott P. Carroll; B: Scott P. Carroll

A: Soapberry bugs in Australia feed by inserting their beak into fruits to reach the seeds inside. In this photo, a soapberry bug feeds on a woolly rambutan fruit, which is native to Australia. B: In the 1960s, an introduced fruit, the balloon vine, became common in eastern Australia. Soapberry bugs started feeding on it, even though it was larger than the native fruit. C: In eastern Australia, the beaks of Australian soapberry bugs that feed on balloon vines have evolved to longer lengths in less than 50 years. (Data from Carroll et al. 2005.)

As one species adapts to another one, its partner may evolve as well. When wasps attack aphids or other hosts, natural selection favors resistance in the hosts—which, in turn, favors counterattacks in the wasps. This transformation does not happen uniformly through each species in one of these partnerships. From one population to the next, the strength of selection may be weak or strong. Some populations may be very small, for example, so that selection is overwhelmed by drift. Other populations of hosts may experience selection on other traits that prevent them from evolving strong defenses.

This complexity is obvious when you look at the geography of coevolution. FIGURE 12.5 shows the interactions between the fly *Drosophila melanogaster* and a species of parasitoid wasp (Kraaijeveld and Godfray 1999). The map on the left shows the percentage of flies in different areas that can encapsulate the wasp eggs. The map on the right shows the percentage of wasps that can resist this encapsulation and grow anyway. The complex pattern emerges in part from the trade-off that the flies face between resisting wasps and competing for food. If a wasp acquires mutations that give it stronger resistance, it may be slower at finding food than more vulnerable wasps are (Fellowes, Kraaijeveld, and Godfray 1998).



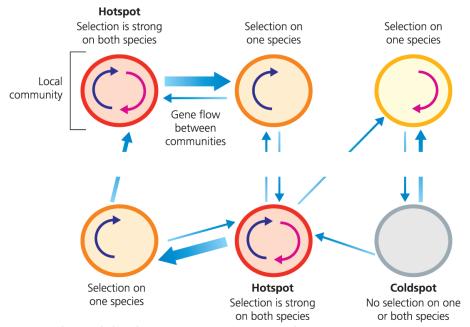
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FIGURE 12.5

Across Europe, *Drosophila melanogaster* flies are parasitized by the wasp *Asobara tabida*. The resistance of the flies (A) and the successful parasitism of the wasps (B) show strong geographic variation. (Data from <u>Kraaijeveld and Godfray 1999</u>.)

The Mosaic of Coevolution

To explain these patterns, John Thompson, an evolutionary biologist at the University of California, Santa Cruz, developed an idea he calls the geographic mosaic theory of coevolution (<u>Thompson 2010</u>). Thompson proposed that there are coevolutionary "hotspots" where partner species are coevolving rapidly, as well as coevolutionary "coldspots" where little coevolution is occurring (<u>FIGURE 12.6</u>). In some parts of their range, they may be precisely adapted to each other in one place, and mismatched in another.



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FIGURE 12.6

The geographic mosaic theory explains coevolution as selection between partners taking place in linked populations. Some populations are coevolutionary "hotspots," where selection is strong on both species. Others are "coldspots," where one or both species experienced no selection. Gene flow delivers coevolved genes from one population to another. (Information from Thompson 2010.)

At any moment, Thompson argued, a map of two coevolutionary partners is a mosaic, made up of many populations in different relationships. What's more, that mosaic changes over time, as genes flow between populations. Thompson's theory predicts that coevolution can produce a variety of relationships, which fall into two categories: antagonistic relationships and mutualistic ones.

The most spectacular form of antagonistic coevolution occurs when one species invests in defenses, spurring its partner to invest in greater counter-defenses, spurring it in turn to invest in even greater defenses. This process is known as a coevolutionary arms race. Thanks to the work of the Brodies and their colleagues, the rough-skinned newt and common garter snake have become one of the best-understood arms races in nature.

After the ability to produce TTX evolved in rough-skinned newts, the Brodies found, resistance to the toxin evolved in common garter snakes. The Brodies and their colleagues have identified some of the mutations in the snakes that alter the shape of the receptor to which TTX normally binds. In populations where common garter snakes can resist TTX and eat the newts, selection then favors newts with even greater production of TTX.

The adaptations that arise during coevolutionary escalation can impose a cost on each species. TTX is a large molecule whose production demands a lot of the salamanders' energy. The more TTX a rough-skinned newt produces, the greater its investment. The Brodies and their colleagues have found that resistance also imposes a cost on snakes. Resistant snakes travel more slowly than susceptible ones, and so they may be less successful at hunting.

The geographic pattern of TTX production in newts and resistance in snakes reflects this combination of escalation and its cost. There's a tremendous range of variation in these two traits (FIGURE 12.7). In some places the newts produce very deadly toxins, and the snakes are highly resistant. In other places the snakes have no resistance to speak of, and the newts produce only barely detectable levels of toxins. The newts and snakes are well matched in their level of toxins and resistance in a few hotspots. But the newts and snakes are also mismatched in about a third of their territory.



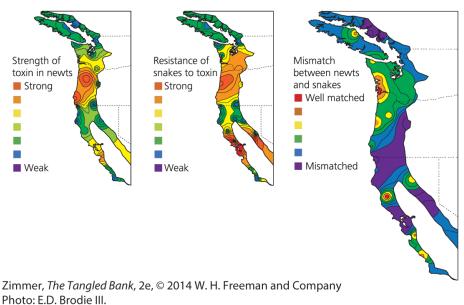


FIGURE 12.7

in some regions, rough-skinned newts and common garter snakes have mismatched levels of toxicity and resistance. The maps show the toxicity of newts (left) and distribution of resistance in snakes (center) where both species coexist. The map at right shows how well matched the two species are across their ranges. Blue and purple regions show coldspots, where mismatch results in little coevolution. Yellow, orange, and red show hotspots, where closely matched toxin and resistance levels reflect coevolutionary escalation. (Information from <u>Brodie 2011</u>.)

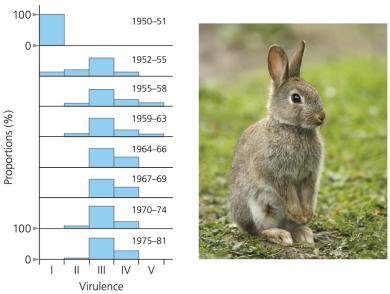
Edmund Brodie III and his colleagues argue that the species become mismatched because it is easier for snakes to evolve resistance than for newts to evolve stronger toxins. It takes just the change of just a single amino acid for a snake's neuron channels to effectively block the newt toxin. On the other hand, a whole series of mutations are necessary for newts to make more deadly toxins. This means that as soon as a snake becomes highly resistant, the arms race is essentially over. No small increase in the strength of a newt's toxins will make the newt any less likely to be eaten (<u>Brodie 2011</u>).

The Coevolution of Disease

Like predators and prey, parasites and hosts can experience antagonistic coevolution. But parasites do not necessarily get trapped in arms races with their hosts. While some parasites are deadly, others impose only a small cost. The Ebola virus almost always causes people to experience a swift, horrific death, for example, while a cold virus can produce billions of descendants inside of us while making us feel under the weather for only a few days.

To understand why pathogens cause different levels of damage—known as virulence—we have to take a look at the pathogens themselves. During an outbreak, many genetically distinct strains are circulating from host to host. Often, a single host is infected with two or more strains at the same time. Highly virulent strains exploit their host more aggressively, reproducing quickly and passing on their alleles to their descendants. The competition within a host will thus select for increased virulence. But virulence is a risky strategy for pathogens, because they may kill their hosts. If they can't spread to new hosts before the old host dies, they will die as well. In other words, selection for transmission to new hosts favors reduced virulence.

The virulence of any particular pathogen emerges from its own trade-off between within-host and between-host selection. And as a pathogen's environment changes, it may evolve to be deadlier or milder than its ancestors. One of the best-documented examples of this kind of attenuated coevolution has unfolded in Australia over the past 60 years (Kerr et al. 2012). In 1859 rabbits were introduced to Australia by Thomas Austin, an immigrant farmer, so that he could have game to shoot. Without predators to control them, the rabbits exploded across the continent, eating so much vegetation that they began to cause serious soil erosion. In the 1950s, scientists deployed a biological counter-offensive, known as rabbit myxoma virus (FIGURE 12.8).



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FIGURE 12.8

Myxoma virus was introduced into Australia to control the exploding rabbit population. This graph shows the virulence of the virus over time, as measured by "virulence grades" (I is the deadliest, V is the mildest). Initially the virus was highly lethal, and later it became less virulent. Less virulent strains were able to spread more effectively than more virulent ones. A similar evolution took place when myxoma virus was introduced to France to control rabbits there. (Data from Begon, Townsend, and Harper 2006.)

The rabbit myxoma virus, which was discovered in South America, causes deadly infections in hares on that continent. The virus was introduced to Australia in the hopes that it would be just as deadly against the imported rabbits there. At first, it lived up to those hopes: scientists found that 99.8 percent of infected Australian rabbits died. But within a few years, the virus's mortality rate dropped to between 70 and 95 percent. That's still fairly deadly, but not deadly enough to keep the fast-breeding rabbits in check. As a result, rabbits continue to be an ecological blight on Australia today because the virulence of the myxoma virus evolved to an intermediate, less deadly state.

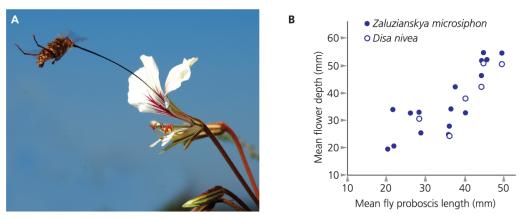
In 1952, the same experiment took place in Europe, where another myxoma virus strain was imported to France to control rabbits. As in

Australia, the virus turned from highly deadly to moderately deadly. The two strains of myxoma acquired different sets of mutations over the past 60 years. But despite their differences, they converged on the same optimal level of virulence.

Mutual Aid

At the other end of the coevolution spectrum are species that benefit one another—a relationship known as mutualism. In mutualisms, an increase in fitness of one species can potentially increase the fitness of its partner. If a mutation results in plants supplying mycorrhizal fungi with more carbon, for example, the plant also benefits because the fungi will be able to grow faster and provide more nutrients from the soil.

As with other forms of coevolution, mutualisms evolve as geographic mosaics. In South Africa, biologists Steven Johnson and Bruce Anderson have documented geographic mosaic in the coevolution of long-tongued flies (*Pro-soeca ganglbaueri*) and the flowers they pollinate. Their "tongue" is actually a tube-like organ, called a proboscis, that they dangle behind them as they fly. When they find a flower they want to drink nectar from, they fold their proboscis forward until it extends before them a distance several times longer than their entire body (**FIGURE 12.9**).



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FIGURE 12.9

A: Long-tongued flies in South Africa feed on flowers with correspondingly long tubes. These extremes are the result of the coevolution of the flies and flowers. B: This graph shows the proboscis length of the fly *P. ganglbaueri* compared to the flowers it feeds on. *Z. microsiphon* is its main source of nectar, while *Disa nivea* is a deceptive orchid that mimics

Z. microsiphon but provides no food. In different populations of the flies and the flowers, different matching lengths have coevolved. (Information from <u>Johnson and Anderson</u> 2010.)

These flies use their extraordinary proboscises to feed on the nectar of long-necked flowers, especially the flowers of a species called *Zaluzianskya microsiphon*. As they try to push their proboscises to the bottom of the deep nectaries, the flies rub their heads on the flowers, picking up pollen. When the flies visit another flower, the pollen from the first plant can fertilize the second one's ovules (<u>Johnson and Anderson 2010</u>).

Anderson and Johnson traveled to 16 sites in South Africa, where they caught hundreds of long-tongued flies. They measured the length of each fly's proboscis, along with many other traits of the flies. At the same 16 sites, they also gathered *Zaluzianskya* flowers, measuring them as well. The scientists found a striking pattern. At some sites, the flies have proboscises as long as 50 millimeters (2 inches), closely matching the depth of the *Zaluzianskya* flower tubes at those sites. At other sites, the flies have proboscises only half that length. Their shorter proboscises are matched by shorter flower tubes.

Anderson and Johnson argue that natural selection must be driving the differences in the length of the proboscis. They suspect that *Zaluzianskya* flowers at some sites dominate the sources of nectar for the flies, resulting in strong pair-wise reciprocal selection between the species. Natural selection favors deeper tubes for nectar in these places, because it forces the flies to pick up pollen as they struggle to reach their proboscises into the tubes. As the flower tubes get deeper, natural selection favors longer proboscises, which in turn selects for deeper flower tubes. But in other populations, the flowers may have to compete with other species with smaller flowers and shorter tubes, thus diluting the strength of reciprocal selection. If a fly can't reach to the bottom of a *Zaluzianskya* flower, it can always drink from a flower of another species, and having too long a proboscis may make this more difficult. At these sites, natural selection may favor flies with shorter proboscises as well as *Zaluzianskya* flowers with shorter tubes.

Coevolution-Fueled Biodiversity

As species coevolve with one another, they also give rise to new species. In 1964, the biologists Peter Raven and Paul Ehrlich argued that coevolution could speed up the rate of diversification (Ehrlich and Raven 1964). In other words, we owe much of the world's biodiversity to coevolution.

As a case study in coevolution, Ehrlich and Raven focused on milkweed plants. There are 130 different species of milkweed (*Asclepias*), and the scientists argued that their diversity was fostered by their coevolution with the insects that feed on them. Milkweeds have many defenses against caterpillars, such as dense hairs that make it hard for the insects to reach their stems. If a caterpillar does manage to pierce a stem, sticky white fluid bursts out of the plant. And even if the insect should still manage to keep eating, the plant produces a cocktail of toxic molecules that can cause the caterpillar serious harm (FIGURE 12.10).



Photo: Anurag Agrawal.

FIGURE 12.10

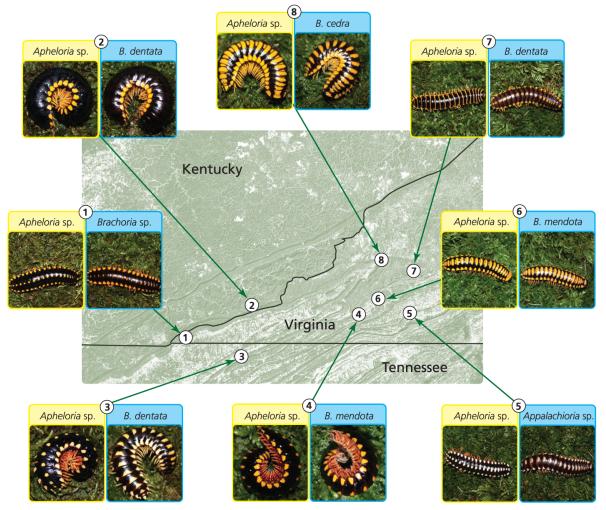
A caterpillar chews on a milkweed plant. In response, the plant's pores release a sticky latex fluid, along with toxic chemicals. Caterpillars, in turn, have evolved defenses against these plant weapons.

Evolving Mimics

Some species of butterflies feed on toxic plants and store the toxins in their tissues. When a bird attacks these insects, it spits them out immediately. Birds are intelligent animals, and as a result, they learn to recognize what toxic butterflies look like and avoid them.

In the 1870s, Fritz Müller, a German naturalist who worked in Brazil, noticed that different toxic species of butterflies often resembled one another. He proposed that the butterflies had converged through coevolution on the same pattern. If the number of individuals with the same appearance increased, unrelated species could all benefit because predators would be even more likely to learn to avoid them.

This phenomenon, which came to be known as Mülle-rian mimicry, turns out to be surprisingly common. in 2009, for example, Paul Marek and Jason Bond of East Carolina University in North Carolina described Müllerian mimicry in seven species of millipedes in the Appalachian Mountains (Marek and Bond 2009). Each of the millipedes produces enough cyanide to kill 18 pigeon-sized birds. Marek and Bond found that at many sites, unrelated pairs of toxic millipede species had evolved similar warning colors (FIGURE 1).



Photos: PNAS Permissions Credit: Adapted from "A Mullerian mimicry ring in Appalachian millipedes" by Marek and Bond. PNAS, 2009: Vol. 106, No. 24, pp 9755–9760.

FIGURE 1 Millipedes in the Appalachian Mountains produce cyanide, making them toxic to birds. Like toxic butterflies, they have evolved Müllerian mimicry. The numbers on this map mark sites where scientists have collected specimens from populations of *Apheloria* (yellow boxes). The blue boxes contain specimens of other species from the same sites.

The caterpillars, meanwhile, have defenses of their own. They can disarm the toxins in the milkweed with special enzymes. They can sabotage the milk defense by cutting holes in the vessels that it flows through.

Raven and Ehrlich argued that the plants and insects evolved these traits in a back-and-forth sequence. In the first step, the milkweed plants evolved a new defense that shielded them from the caterpillars. They were able to grow fast and produce many seeds. They spread and diversified into new species. Over time, counter-defenses evolved in the insects, allowing them to exploit the new milkweed species. Now the insects were the ones to enjoy an explosion of resources. Their numbers increased, and they evolved into new species. Then it was the milkweed's turn, and so on, in a cycle of diversification.

Generations of scientists have investigated this "escape-and-radiate" model, and a number of studies have supported it not just for milkweed plants, but for other plants and insects (<u>Janz 2011</u>). But this coevolution is different than the intimate species-to-species partnership found in, say, long-tongued flies and their flowers. Instead, many species of insects and many species of plants interact with each other. This loose interaction is called diffuse coevolution.

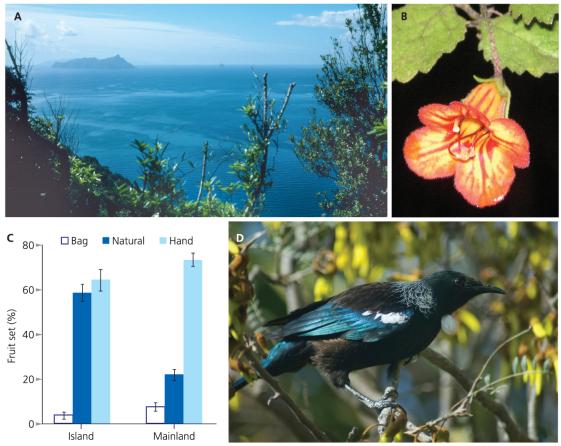
While Raven and Ehrlich saw antagonistic relationships as an engine for coevolution, mutualism is also turning out to be powerful in some cases. Szabolcs Lengyel, a biologist at North Carolina State University, and his colleagues recently took a look at the diversity that arises from the coevolution of flowering plants and the ants that spread their seeds (Lengyel et al. 2009). About 11,000 known plant species around the world grow fleshy handles on their seeds called elaiosomes. After ants bring the seeds to their nests, they eat the elaiosomes and discard the seeds in a special room in their colony. There the seeds sprout, protected from being eaten by other animals.

Elaiosomes have evolved independently at least 101 times, as Lengyel and his colleagues reported in 2009. They also found that ant-dispersing lineages contain over twice as many species as the most closely related lineages of plants. Ants may foster this plant diversity by protecting the seeds. The plants end up growing in a small range around specific ant colonies, causing them to become reproductively isolated.

Coevolution fosters biodiversity, but it may play a role in extinction as well (<u>Toby Kiers et al. 2010</u>). If one species depends on another one for its survival, then it will not be able to endure after the other species becomes extinct. If it can shift to a new partner, however, it may be able to survive. Mass extinctions in the past offer some clues as to how coevolution makes species vulnerable. Some species of corals live mutualistically with algae

and some do not. In the last major mass extinctions, 65 million years ago, mutualistic corals suffered about four times more extinctions than non-mutualistic corals. These extinctions coincided with a huge asteroid impact that blocked out the light of the sun for months. It's possible that this blackout killed off photosynthetic algae as well as the corals that depended on them for survival (<u>Kiessling and Baron-Szabo 2004</u>).

Scientists are investigating the role that coevolution plays in the current extinction crisis, which we surveyed in the last chapter. Sandra Anderson of the University of Auckland and her colleagues, for example, are studying the effect of the introduction of cats and other mammal predators to New Zealand (Anderson et al. 2011). These introduced predators have helped to drive 49 percent of New Zealand's native bird species extinct. Anderson and her colleagues wondered what happened to the plants that depended on the extinct birds for pollination (FIGURE 12.11).



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FIGURE 12.11

A: New Zealand and its surrounding islands are home to many endemic species, such as the flower *Rhabdothamnus solandri* (B). For pollination, many of these plants depend on native species of birds such as the tui (*Prosthermadera novaeseelandiae*), shown in D. Many native New Zealand birds are now endangered or extinct, raising the question of how their disappearance will affect plants. C: New Zealand researchers gauged the effect of bird extinctions by measuring how much fruit *R. solandri* produced. They studied a mainland population that has lost two of its three main pollinators and compared it to flowers on islands where all three birds are still present. In one trial, they put bags over the flowers. In another, they pollinated the flowers by hand. In the third trial, they measured the fruit set produced by natural pollination. The scientists found that mainland flowers produce much less fruit due to the local extinction of pollinators. (Data from Anderson et al. 2011.)

To find out, they studied the plants on the upper portion of the North Island, comparing them to the plants on the small adjacent islands. On the mainland, two of the three native pollinating birds have become extinct. But on the nearby islands, all three of those pollinators—the bellbird, stitchbird, and tui—still survive. The scientists hypothesized that the two populations of plants had different levels of reproductive success.

They tested this hypothesis on the native flower, *Rhabdothamnus* solandri, which grows on both the mainland and the islands. They placed bags over some flowers so that they could not be pollinated at all. They pollinated other plants by hand. And they left a third group of the plants to be pollinated naturally by birds. On both the mainland and the islands, Anderson found, bagged flowers produced almost no fruit at all. Plants pollinated by hand produced equally abundant fruits in both sites. But the scientists discovered a big difference between the islands and mainland when they examined the naturally pollinated flowers. The island flowers produced about as much fruit on their own as they did when pollinated by hand. The mainland flowers, by contrast, produced far fewer.

These results suggest that native flowers on the mainland of New Zealand are suffering pollination failure because they have lost their pollinators. Because *R. solandri* is a slow-growing plant, it may take a long time for it to

disappear from the mainland. But without its coevolutionary partners, it may be doomed.

How Two Species Become One

The glassy-winged sharpshooter is one of the most dreaded agricultural pests in the United States (FIGURE 12.12). It pushes its needle-like mouthparts into trees and other plants and then drinks the xylem in the woody fibers. As it feeds, the sharpshooter can deliver a number of species of pathogenic bacteria that can kill plants. Entire wine vineyards can be destroyed in a few years by Pierce's disease, which is caused by bacteria transmitted by sharpshooters.



Photo: Clarence Holmes Wildlife/Alamy.

FIGURE 12.12

Sharpshooters have coevolved with bacteria that supply the insects with essential nutrients. The bacteria benefit by getting food and shelter from the insects. The sharpshooters even develop special organs called bacteriomes (the orange spots on the sides of the insect).

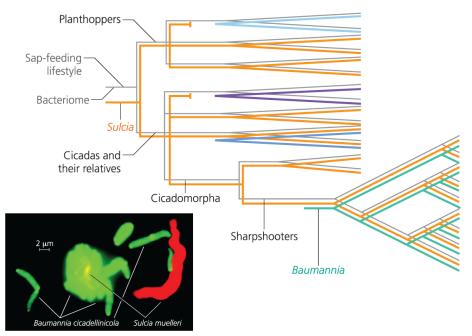
As devastating as glassy-winged sharpshooters may be, they are also metabolic marvels. Xylem fluid is a poor source of nutrition for animals because it lacks many essential nutrients. Sharpshooters must drink huge amounts of the fluid, much of which they excrete in little droplets, creating a rain-like shower. On their own, the sharpshooters would not be able to survive on the concentrated fluid they consume. They depend on several species of xylem-feeding bacteria that live in a pair of special organs called bacteriomes, which look like a pair of orange spots on the back end of the insect (Figure 12.12). The cells in the bacteriomes can house huge numbers

of the bacteria, which the insects then pass down to their offspring (Moran 2007).

Mutualists like these bacteria, which must live inside coevolutionary partners, are known as endosymbionts. Endosymbionts are intimately involved in the biology of many animals and plants. We humans are home to 100 trillion bacteria and archaea, belonging to several thousand species. Some of these microbes produce enzymes that we lack, allowing us to digest tough plant matter. Some species synthesize vitamins, while others defend us against pathogenic species.

The evolutionary dynamics of endosymbionts differs in some important ways from the dynamics of free-living mutualists. For example, they have the potential to enter into long-lasting relationships that may stretch over millions or even billions of years. One way to reconstruct this long history is to compare the phylogenies of endosymbionts and their hosts.

Nancy Moran, an evolutionary biologist now at the University of Texas, has used this method to trace the evolution of the endosymbionts inside the glassy-winged sharpshooter. One of these endosymbionts is called *Sulcia*. Moran and her colleagues have compared *Sulcia* in sharpshooters to related bacteria living in other sap-feeding insects with bacteriomes. The pattern of the phylogeny of the *Sulcia* strains matches the phylogeny of their insect hosts almost perfectly (FIGURE 12.13). *Sulcia* have been cospeciating with their hosts since their common ancestors first acquired the bacteria. Judging from the earliest fossils of the insect hosts, this mutualism must have arisen 270 million years ago at the latest (Moran 2007).



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: From Symbiosis as an adaptive process and source of phenotypic complexity by Nancy A. Moran. PNAS, 2007: Vol. 104, No. Suppl. 1 8627–8633, © National Academy of Sciences, U.S.A.

FIGURE 12.13

Sharpshooters rely for their survival on a collection of bacteria, including *Sulcia* and *Baumannia* (shown here in a micrograph). Studies on the DNA of the insects and their bacteria have revealed that some 270 million years ago, *Sulcia* was already living inside the sapfeeding ancestor of sharpshooters and related insects. In some lineages, *Sulcia* was lost and new bacteria came to take its place (light blue, purple, and dark blue branches). In the more recent ancestors of sharpshooters, *Baumannia* joined *Sulcia* inside the insects, thus permitting their hosts to feed on xylem, a very poor source of nutrition. (Information from Moran 2007.)

The lineage that produced today's thousands of sharpshooter species evolved roughly 50 million years ago, judging by the oldest sharpshooter fossils. The early sharpshooters shifted from other plant fluids to xylem sap. They also became hosts to a new mutualist, a bacterium called *Baumannia cicadellinicola*. Once *Baumannia* became an endosymbiont, it cospeciated with sharpshooters along with *Sulcia*.

When endosymbionts begin to be passed down from parents to offspring, they undergo some of the most extreme forms of coevolution ever

documented. They always have a rich supply of nutrients on hand, which they no longer need to produce. Mutations that disable the genes involved in being a free-living microbe no longer lower their fitness. As a result, the genomes of many endosymbionts have shrunk dramatically. *Tremblaya*, a species of bacteria that lives in mealybugs, currently has the record for the smallest known genome. It has only 130,000 base pairs of DNA. The human genome is over 10,000 times bigger (McCutcheon and von Dohlen 2011).

Both *Sulcia* and *Baumannia* also have evolved small genomes, and they've done so in a particularly elegant way. *Baumannia* has lost all of its genes for synthesizing amino acids except for histidine. The main service *Baumannia* provides for its sharpshooter host is synthesizing vitamins and other nutrients. *Sulcia*, on the other hand, has retained all of its genes for amino acids—except for histidine. Together, the two bacteria species can provide a sharpshooter with all of the amino acids it needs to stay alive. In other words, these two microbes have not only coevolved with their insect host, they have coevolved with each other (McCutcheon and Moran 2010).

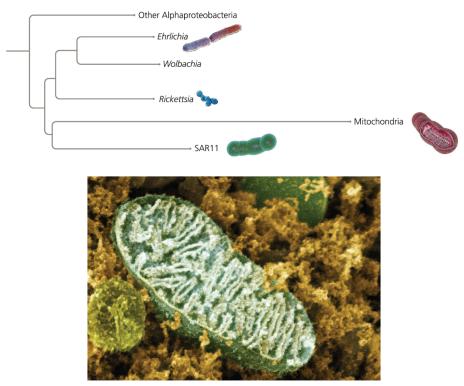
Our ancestors also acquired endosymbionts, which we depend on today for our survival. They have become so well integrated into the eukaryote cell that generations of cell biologists didn't recognize them as the descendants of free-living bacteria. They are mitochondria, the sausage-shaped structures that use oxygen, sugar, and other molecules to produce energy for eukaryotic cells. Mitochondria also carry out other important jobs, such as building clusters of iron and sulfur atoms that are then attached to certain proteins.

Mitochondria have puzzled cell biologists ever since their discovery in the late 1800s. They seem very much like little cells within our cells. They are surrounded by two membranes and carry their own DNA, which replicates as they divide. At the dawn of the twentieth century, Russian biologists proposed a solution to the mystery: mitochondria were once free-living, oxygen-consuming bacteria that entered a single-celled host (Sapp 1994). That host was an early eukaryote, and its descendants today include animals, plants, fungi, and protozoans.

The proposal was generally forgotten until the 1960s, when Lynn Margulis, a biologist at the University of Massachusetts, revived it. In the 1970s, scientists were able to test her hypothesis by examining bits of mitochondrial DNA. This DNA did not closely resemble any human genes,

any genes of any animals, or even any eukaryotes. The closest matches came from bacteria.

Since then, scientists have zeroed in on the particular kind of bacteria that mitochondria evolved from. The most recent studies (<u>Thrash et al. 2011</u>) identify a clade of bacteria known as SAR11 as the closest living relatives of mitochondria (<u>FIGURE 12.14</u>). SAR11 is an extremely abundant clade of marine bacteria; about 25 percent of all bacteria in the ocean belong to it. These bacteria are aerobes, using oxygen to feed on dissolved organic carbon.



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo: Dr. David Furness, Keele University/Science Source.

FIGURE 12.14

Mitochondria generate energy for our cells. These sausage-shaped structures were originally free-living bacteria that were later engulfed in our single-celled ancestors. They are now present in the cells of most eukaryotes. This evolutionary tree shows the relationship of mitochondria to their closest bacterial relatives, a lineage of ocean microbes. (Information from <u>Thrash et al. 2011</u>.)

For many years, scientists were divided about when exactly eukaryotes acquired the first mitochondrion. Some argued that this transition occurred well after the origin of eukaryotes, pointing to the absence of mitochondria in some single-celled protozoans such as *Giardia lamblia*, a protozoan that can cause painful diarrhea. It appeared that only after *Giardia* and other mitochondria-free eukaryotes branched off did bacteria become mitochondria.

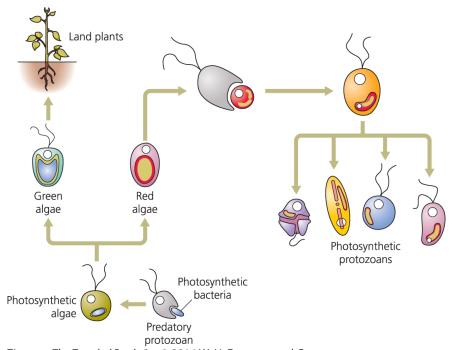
But most experts now reject this hypothesis based on recent research on eukaryotes, including studies on *Giardia*. In 2003, Jorge Tovar, of Royal Holloway College in England, and his colleagues discovered proteins in *Giardia* that were very similar to the proteins in mitochondria that build iron and sulfur compounds. The scientists manipulated the proteins so that they would light up inside *Giardia*. It turned out that the proteins all clumped together in a tiny sac that had, until then, gone unnoticed (<u>Tovar et al. 2003</u>).

Tovar and his colleagues proposed that these sacs, which they dubbed mitosomes, are vestiges of full-blown mitochondria. As *Giardia* adapted to an oxygen-free life in the intestines of animals, it lost its ability to use oxygen and its mitochondria evolved into mitosomes. Similar results have emerged from other supposedly mitochondria-free eukaryotes. They have genes, proteins, and compartments that all show signs of being remnants of full-blown mitochondria.

The most compelling hypothesis that accounts for these results is that oxygen-consuming, SAR11-like bacteria took up residence inside some of the first eukaryotes some 2 billion years ago. Judging from the fact that single-celled eukaryotes today are loaded with bacteria, such an infection would have been routine. Early eukaryotes exploited the energy provided by their new resident, and they gradually abandoned their own energy-generating proteins. From those early hosts, all the lineages of living eukaryotes evolved.

Mitochondria today are not typical bacteria by any measure. They would not survive for a second outside cells. That's because they've been evolving inside the cells of eukaryotes for billions of years. As they replicated inside their host cells, their DNA mutated. Some of those mutations deleted genes that were no longer necessary to their survival, now that they were protected inside another cell. Other mutations caused genes to be transferred from the mitochondria to the DNA in the nucleus of the cell.

Another coevolutionary merger took place hundreds of millions of years later, in the ancestors of plants (FIGURE 12.15). Plants descended from single-celled eukaryotes that originally were most likely microscopic predators, feasting on bacteria. At some point, however, they became hosts to bacteria that could carry out photosynthesis. At first they may have gotten energy both from eating other organisms and from capturing sunlight. Some photosynthetic eukaryotes live this way today in the ocean. But the ancestors of plants gradually shifted over to rely entirely on photosynthesis. Along with their mitochondria, they carry remnants of the photosynthetic bacteria, called plastids. Every patch of living green you see, from a blade of grass to a forest of redwoods, got its start with a merger of two species (Gross and Bhattacharya 2009).



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FIGURE 12.15

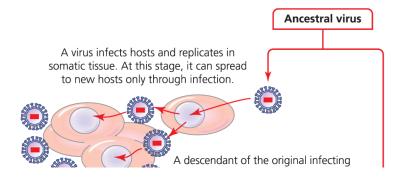
Plants can use sunlight to grow with the help of bacterial partners. *Lower left:* A single-celled ancestor of plants engulfed photosynthetic bacteria. The descendants of these photosynthetic cells evolved into several living lineages, including red algae and green algae (*upper left*). one lineage of green algae evolved into land plants. *Upper right:* One lineage of red algae was engulfed by another host, which evolved into many lineages of living photosynthetic protozoans. (Information from <u>Bhattacharya et al. 2007</u>.)

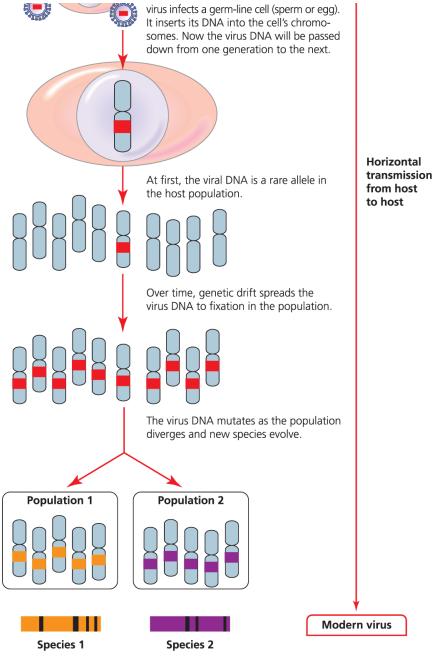
Invasion of the Genomic Parasites

In 2006, Thierry Heidmann, a researcher at the Gustave Roussy Institute in Villejuif, France, resurrected a virus that had been dead for millions of years. Heidmann and his colleagues did not discover the virus buried in ice or hidden in a cave. They found it in the human genome. All human beings on Earth carry remnants of the virus's genetic sequence in their own DNA. This spectacular revival has helped scientists to understand one of the strangest yet most important forms of coevolution: the coevolution that takes place between different parts of our genome.

The virus that Heidmann revived belongs to a group known as retroviruses. They infect their hosts by creating an RNA copy of their genome, which is then turned into DNA that is inserted into the genome of a host cell. Typically, these embedded viruses hijack the biochemistry of their host, using it to produce new viruses that then burst out of the cell. But scientists have also discovered retrovirus-like DNA that is a permanent part of the human genome, passed down from one generation to the next.

It's likely that these virus-like stretches of DNA descend from retroviruses that infected sperm or egg cells. An organism produced from one of those infected sex cells carried the virus in all the cells of its body, including its own sex cells. Over the course of many generations, mutations to this viral DNA robbed the viruses of their ability to make new viruses that could escape their host. The best they could manage was to make copies of themselves that could be inserted back into the same cell's DNA. Eventually even that trait was lost, and the virus's DNA became inert (FIGURE 12.16).





Scientists can identify variants of the original virus in the chromosomal DNA of different host species.

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FIGURE 12.16

Endogenous retroviruses sometimes become incorporated into host genomes. They can be recognized in host genomes by comparing them to related viruses that still infect hosts. (Information from <u>Johnson 2010</u>.)

To test this idea, Heidmann tried to revive one of these retroviruses into its once-active form. He and his colleagues selected a virus-like segment of DNA found only in humans. They found slightly different versions of the segment in different people. These differences presumably arose as the original retrovirus mutated in different lineages of humans. Heidmann and his colleagues compared the variants to determine what the original sequence had been. They built a piece of DNA that matched the original sequence and inserted it into a colony of human cells reared in a petri dish. Some of the cells produced new viruses that could infect other cells. Heidmann named the virus Phoenix, for the mythical bird that rose from its own ashes (Dewannieux et al. 2006).

Phoenix has been found only in humans, which indicates that the virus infected our ancestors after they had branched off from the apes some 7 million years ago. But we do share other endogenous retroviruses with other apes, as well as with monkeys in Africa and Asia. These viruses must be much older because the common ancestor of all these primates lived about 30 million years ago. After the ancestors of those viruses infected early primates, they continued to replicate and to insert new copies back into the genomes of their hosts. In your own genome, making up about 8 percent of your DNA, there are almost 100,000 fragments of endogenous retroviral DNA. They take up about four times more of the human genome than the 20,000 genes that encode proteins (<u>Johnson 2010</u>).

Endogenous retroviruses were not the first "jumping genes" scientists discovered in the genome. In the 1950s, biologist Barbara McClintock was studying the genes that control the color of corn kernels. She discovered that the genes could make a copy of themselves, which could be inserted elsewhere within the corn genome from one generation to the next. In 1983, she was awarded the Nobel Prize for her work.

Later generations of scientists discovered a vast menagerie of DNA elements that can move through the genome. About half of the human genome is made up of these mobile genetic elements, which number in the millions. While most mobile elements are "dead"—that is, they cannot replicate themselves—a few of them still do. One out of every 20 to 100 human babies acquires a new insertion of a mobile element.

It appears that at least some of these mobile genetic elements got their start as retroviruses. Mutations deleted much of their original DNA, leaving

behind just the bare minimum instructions for making new copies of themselves that could then be reinserted into the host genome. Other mobile elements may have entered the genomes of our ancestors by hitchhiking with viruses from other species.

Endogenous retroviruses and other mobile genetic elements are, in effect, genomic parasites. As they spread within a host genome and hop to new hosts, they can harm their hosts. Mobile elements that insert themselves into new places in the genome can disrupt a cell's normal rhythms of growth and division. A cell may begin to multiply out of control, giving rise to cancer. If a mobile element inserts itself in the middle of an essential gene, a cell may no longer be able to produce a vital protein. Experiments on mobile genetic elements in *Drosophila melanogaster* have allowed scientists to measure their effect on fitness. Flies with two copies of a mobile element called mariner lived for 57.6 days on average, while mariner-free flies lived for 61.4 days. In other words, the mariner mobile element cuts short the life of its host.

By lowering the fitness of their hosts, mobile genetic elements and endogenous retroviruses lower their own fitness because fewer hosts will be around to carry them. But many of these genomic parasites can replicate themselves so quickly that they can still spread through a population despite the harm they cause. Mobile elements demonstrate how selection can take place at several levels at once. A mutation can raise the fitness of a mobile genetic element, even as it lowers the fitness of the organism that carries it.

The coevolution of genomes and genomic parasites is as complex as the coevolution of two free-living species. The genomic parasites evolve ways to spread themselves, and host genes evolve ways to halt them. Sometimes DNA that originally behaved like a parasite takes on a new function that benefits the host. These "domesticated" parasites blur the line between coevolutionary partners even more than mitochondria and plastids do.

Ironically, one of these domesticated parasites helps us to fight off other parasites. The immune systems of sharks, bony fishes, and all land vertebrates are able to recognize a vast number of pathogens. They do so thanks to a set of genes that can produce receptors and antibodies with an equally vast number of different shapes. To generate these molecules, our immune cells must cut apart the corresponding genes and then join them

together. Depending on what gets cut out, the genes produce molecules of different shapes.

The genes that encode the cutting proteins are called *Rag1* and *Rag2*. In 2005, Vladimir Kapitonov and Jerzy Jurka, two geneticists at the Genetic Information Research Institute in Mountain View, California, discovered that the two genes were significantly similar to a family of mobile genetic elements called Transib. Transib mobile elements don't just resemble *Rag1* and *Rag2* in their DNA sequence. They also cut and paste DNA in the same way. Kapitonov and Jurka argue that in some ancient fish that lived 500 million years ago, Transib mobile elements mutated so that they began to cut and paste immune system genes. Now, instead of being a burden to their hosts, they help their hosts fight off disease (<u>Kapitonov and Jurka 2005</u>).

When Darwin first recognized coevolution, he saw its effects on separate partners—on flowers and bees, for example, or on predators and their prey. Today, however, scientists can see coevolution's marks within us. Our genome has emerged out of a coevolutionary history of cooperation and conflict (Burt and Trivers 2006).

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Minds and Microbes

13

The Evolution of Behavior



Gavin Hunt.

New Caledonian crows (*Corvus moneduloides*) can fashion sticks into tools they use when fishing for insect larvae in tree crevices.

New Caledonia is a remote archipelago lying 1200 kilometers east of Australia. It's also one of the world's great havens of biodiversity. Out of the 3270 species of plants that grow on New Caledonia, for example, 2432 of them are found nowhere else on Earth. Evolutionary biologists from all over the world come to New Caledonia to study its extraordinary inhabitants. One of these scientists is Alex Taylor, who first came to New Caledonia in 2004 while earning his PhD in psychology from the University of Auckland in New Zealand. But Taylor's mission was somewhat different from those of his fellow scientists. He came not to study the island's extraordinary biodiversity; he came to study an extraordinary kind of behavior. All species have their own kinds of behavior—in other words, they have a tendency to react in a particular fashion to a particular situation or stimulus. But on New Caledonia, there are crows with a remarkable behavior: they can make tools.



Brenna Knaebe.

FIGURE 13.1

Alex Taylor has discovered that New Caledonian crows can use tools to get other tools to reach food, suggesting these birds have a capacity for abstract thought.

For a long time, it seemed as if humans were the only animals that could fashion their own tools. This exceptional skill seemed to explain our remarkable success as a species. But in the 1960s, primatologists challenged this notion with reports that chimpanzees used tools as well. They smashed nuts with rocks and fashioned sticks for fishing termites out of nests. Perhaps, the scientists suggested, tool use had evolved in the common ancestors of the great apes and humans, finally reaching full flower in our species. And so it was with great surprise that Gavin Hunt, a scientist at the University of Auckland, discovered in 1992 that New Caledonian crows can fashion tools every bit as impressive as those of chimpanzees. Hunt's discovery raised the possibility that toolmaking had evolved at least a few times in the animal kingdom.

New Caledonian crows eat insect larvae nestled away in the crevices of trees. This kind of diet poses a quandary for them, because their beaks are too short to reach the grubs. They can't hammer their way to the grubs the way woodpeckers do, because their skulls are too weak. Hunt discovered that the birds solve this conundrum by making a tool to grab the larvae. They search for a plant with a suitable branch, which they then pry off. Next, they use their beaks to strip away any small branches and curl the end to fashion a hook. The crows can then dip the hook into the crevice and draw out the larvae.

But twig hooks are not the only tools in the crows' toolkit. They also pluck leaves from pandan plants and snip off the edges until they look like locksmith picks. The crows can then dip these leaf

picks into crevices in trees. When they draw the leaves back out, they also bring out insects snagged on the jagged edges.

Hunt and his colleagues have observed that the crows take good care of their tools. The birds hold onto them with their feet as they eat larvae. They store their hooks and picks on tree perches when they fly to distant hunting grounds.

Intrigued by Hunt's work, Taylor decided to study the crows for his dissertation. He wondered what sort of mental activity went on in the birds as they made their tools. Did they use an abstract representation of the tools in their brains as a guide, or did they simply tear up branches and leaves, following a simple set of rules? To find out, Taylor designed an experiment in which New Caledonian crows could get food only if they could figure out how to use one tool to get another tool.

Taylor put a piece of meat at the far end of a narrow, transparent box. The crows could see the food but not reach it. Near the food box was a second box with a long stick inside. Taylor put bars on the box so that the crow couldn't reach the long stick with its beak. Finally, Taylor tied a short stick to a piece of string, so that it dangled nearby (FIGURE 13.2).

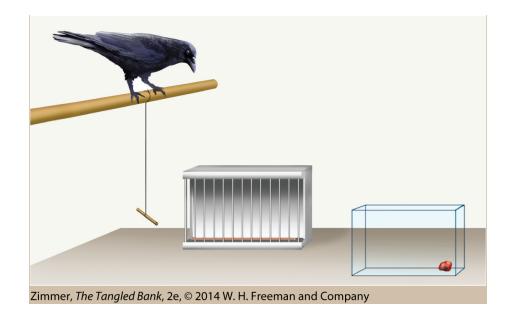


FIGURE 13.2

In Taylor's experiment, food was placed at the end of a transparent box, too far for the birds to reach with their bills. A stick of sufficient length was placed in a cage. The birds figured out they would first have to untie a short stick and then use it to push out the long one. (Information from Taylor et al. 2010.)

The crows inspected the boxes and the sticks and came up with a solution. They hopped to the string and used it to pull up the short stick. Then they pulled the short stick free and used it to fish out the long stick. Finally, the crows used the long stick to reach the food. In other words, New Caledonian crows do not simply see sticks as food hooks. Instead, they invent new solutions to new problems with the tools they have at hand—or, rather, at beak (Taylor et al. 2010).

Making tools is part of the New Caledonian crow's phenotype. Its ability to craft hooks allows it to fish out food that would otherwise be impossible to reach. The energy and nutrients they get from that food then raise their chances of surviving and of having offspring. Thus, to understand the evolution of a species like the New Caledonian crow, we must understand how its behavior evolves.

In this chapter, we investigate the evolution of behavior in a species ranging from microbes to insects to birds to dolphins. To study behavior, scientists develop theories and mathematical models, which generate hypotheses that they can test. They also investigate the biological underpinnings that make behavior possible but also constrain its scope—from networks of signaling molecules in microbes to brains in animals. We'll look at the kinds of behavior that organisms have evolved, including how they interact with members of their own species. We'll examine some of the leading questions about the evolution of social behavior, such as why so many organisms cooperate and even behave altruistically. Finally, we'll look at how scientists search for underlying principles of evolution by comparing different species.

As we'll see, the evolution of tool-using crows on a remote Pacific island can offer hints to our own evolution—which will be the subject of the next chapter.

Behavior Evolves

For a trait to experience natural selection, it must meet three requirements. First, a population must have variation in the trait. At least some of that variation must be due to genetic variation that can be passed down from parents to offspring. And that genetic variation must have an effect on the fitness of an organism. Behaviors meet all three of these requirements.

An extraordinary experiment that has taken place over the past five decades in Siberia shows how a trait can evolve. In the 1959, the Russian biologist Dmitry K. Belyaev started a long-term experiment on fox behavior at the Siberian Department of the Soviet Academy of Sciences (<u>Trut 1999</u>). Belyaev was interested in how wild animals had become domesticated. To replicate the domestication of dogs, Belyaev launched a selection experiment with silver foxes.

Belyaev and his colleagues started by putting 130 silver foxes in cages and running a series of simple tests on them (FIGURE 13.3). When a fox pup reached a month old, a scientist would approach its cage with food in hand. The pup might run away, snarl, or approach the human. The scientist then tried to stroke the fox, which might respond by biting, backing away, or tolerating the contact. Belyaev and his colleagues repeated these experiments until the foxes reached maturity, and then they gave each animal a tameness score. All the foxes were still wild animals, but some were less fearful of humans than others.



RIA Novosti/Science Source.

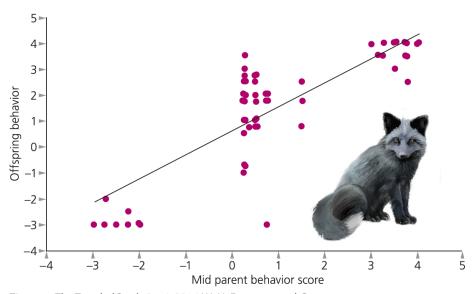
Russian biologist Dmitry K. Belyaev launched an experiment on the evolution of behavior in 1959 that still runs today. He and his colleagues selected foxes to breed based on tameness. Later generations became as affectionate as puppies. Here Belyaev poses with some of his tamed foxes.

Belyaev suspected that the domestication of wolves into dogs started when natural selection favored wolves that didn't respond aggressively to humans. He imposed a similar kind of selection on the foxes. Belyaev and his colleagues picked out the highest-scoring animals and let them mate with one another. The foxes produced a new generation of pups, and the scientists scored them as well. When the pups grew up, the tamest ones in that generation were selected for mating. Belyaev continued the experiment for the next 26 years, till his death in 1985, and other Russian scientists have continued it to this day.

Over the generations, the scientists found that the average tameness score went up. Eventually the foxes underwent an astonishing transformation. Today, they are as friendly as dogs, running up to humans even when no food is on offer. Along with this change in behavior have come many other changes. The foxes play with each other as adults and even wag their tails

like dogs. Their physical appearance has changed too: their coats developed mixes of black and white patches, and their ears grew floppy.

Belyaev's experiment worked because the behavior of the foxes is partly determined by their genes. To demonstrate this, the Russian scientists compared the tameness score of a fox to those of its offspring (<u>Kukekova et al. 2006</u>). As they predicted, the tameness of the parent foxes was a good predictor of the score of their offspring (<u>FIGURE 13.4</u>). If tameness has been only the product of the environment in which Belyaev reared the foxes, there should have been no correlation in the fox families.



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FIGURE 13.4

Researchers compared the tameness of Balyaev's foxes to that of their offspring. As this graph shows, there was a significant correlation between the parent and offspring, indicating that their behavior was influenced by genes. (Information from Kukekova et al. 2006.)

To find some of the genes that influenced the foxes' behavior, the scientists ran breeding experiments similar to those that Hopi Hoekstra and her colleagues ran on oldfield mice to find genes that controlled the color of their coats (page 148). As they bred foxes for tameness, the scientists had also bred a separate population of foxes for wildness, picking out only the

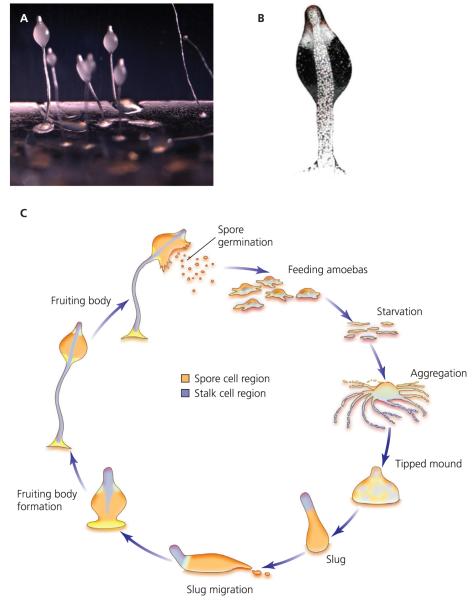
lowest-scoring foxes to mate. They soon ended up with a nasty collection of foxes.

The scientists bred the two groups of foxes to produce hybrids. Then, they searched for segments of DNA that tended to turn up in foxes with the same types of behavior. This stage of the research is still in its infancy, but already it has yielded some tantalizing results. The Russian scientists identified one narrow segment of DNA on chromosome 12 that is particularly strongly linked to friendly or hostile behavior. That same segment has also displayed a strong difference between wolves and dogs. The foxes, in other words, may be repeating the evolution of dogs down at the molecular level (Kukekova et al. 2012).

Behavior without a Brain

It's not too hard to recognize that foxes have a distinctive type of behavior and to see how it evolves. But it can be surprising to discover that behavior is not limited to animals with brains. Even single-celled organisms have distinctive behaviors, and they can be remarkably complex.

Dig up a scoop of forest soil, and that scoop likely contains thousands of microbes called *Dictyostelium discoides* (commonly known as cellular slime mold). This single-celled eukaryote spends most of its existence as a predator, hunting for bacteria, and dividing in two to reproduce. Eventually, the slime molds exhaust their food supply and face the threat of starving to death. They respond by releasing a chemical into the soil, which other slime molds can detect. The slime molds follow this signal to find each other. They swarm into a giant blob, which then stretches out into a slug made up of about 20,000 cells. The cells work together to move as a single unit, crawling through the soil faster than the cells could on their own (FIGURE 13.5).



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company (A): Owen Gilbert.; (B): Genetics Society of America.

A: Dictyostelum discoides, also known as cellular slime molds, are single-celled eukaryotes that display complex behavior. To cope with starvation, individual slime molds converge to form a slug, which then grows into a stalk with a patch of spores on top during reproduction. B: The stalk cells support the spores, but die in the process. Kin recognition has evolved in Dictyostelium as a way of overcoming cheating, so only relatives are included in slug formation. C: The life cycle of Dictyostelium discoideum. (Information from Schaap 2007.)

The cells that make up the slug take on different behaviors, each of which benefits the entire group. Some start to move from the tip to the tail of the slug and back, hunting for bacteria that may sicken the other cells. These police cells drop away from the slug, taking the bacteria with them. The slug moves on, leaving them to starve. Eventually, the slug comes to a halt. The cells at the leading tip—about 20 percent of the entire slug—form an erect stalk by making cellulose and other stiffening molecules. This is a profoundly selfless act, because these molecules are toxic and kill those cells. The remaining 80 percent of the slug cells crawl up the stalk, forming a bulb known as the fruiting body. Inside the fruiting body, cells become hard-coated spores that can be spread by water, wind, or animals. Once they land in a new patch of soil, they revive and begin hunting again for bacteria (Bonner 1967; Strassmann 2010).

As scientists continue to study the microbial world, they are discovering even more of these complex behaviors. It's now abundantly clear that there was a rich diversity of behaviors on Earth over 3.5 billion years ago. And as scientists study the behavior of living microbes, they're discovering some of the patterns by which that behavior evolves.

As with silver foxes, genes play an important part in the behavior of *Dictyostelium*. When these genes mutate, the behavior of the slime molds can change. In 2000, for example, Richard Kessin of Columbia University and his colleagues discovered that slime molds can become cheaters (<u>Dao</u>, <u>Kessin, and Ennis 2000</u>). The scientists isolated a mutant strain of *Dictyostelium* that could form a slug, but failed to form a fruiting body. They found that this strain had acquired a mutation on a gene they dubbed *chtA*. But when Kessin and his colleagues mixed the *chtA* mutants with ordinary slime molds, they found a different result. The two strains of slime molds mixed together in slugs, which could then make a fruiting body. And when the scientists looked inside the fruiting body, they discovered a higher proportion of *chtA* cells than in the starting mixture.

This was a startling result, because only the cells that become spores can survive. In other words, cells with the *chtA* mutation are more likely to survive than those without. To see the effect of this fitness over the long term, Kessin and his colleagues carried out an experiment. It was much like the

experiment that we examined on <u>page 136</u>, in which Richard Lenski and his colleagues allowed *E. coli* to evolve. Kessin and his colleagues started their experiment with a single *chtA* mutant and 1000 regular *Dictyostelium*. They allowed the slime molds to produce slugs and then used the spores in the fruiting bodies to produce the next generation. After just 12 cycles, all of the spores in the fruiting bodies were *chtA*.

The success of cheating in experiments with *Dictyostelium* raises a fascinating question: if cheating can be such a successful strategy, then how can slime molds continue to be so cooperative? It's a question that applies not just to the behavior of single-celled microbes but also to complex, multicellular animals. Later in this chapter, we'll look at the factors that affect the evolution of cooperation and cheating.

Plants Behave, Too

Charles Darwin may not have had the tools to observe microbes behaving, but he was still keenly aware that animals are not the only organisms to evolve behaviors. In fact, he was the first scientist to demonstrate that plants behave, too (<u>Kutschera and Niklas 2009</u>).

Plants can sense many features of their environment—such as light, heat, and gravity—and they respond to that information by changing the way they move and grow. If a plant senses another plant beginning to overshadow it, for example, it will speed up its growth to outstrip its rival. Plants use their sense of gravity to send their roots down instead of up. If the roots encounter an obstacle like a rock, touch-sensitive receptors trigger signals that cause the root to grow around it.

To defend themselves against herbivores, plants behave in remarkable ways. If they sense that a caterpillar is nibbling on their leaves, they may produce toxins to sicken it. They also release chemical signals known as pheromones that cause other plants nearby to strengthen their defenses as well, even before they're attacked. Parasitic wasps that kill plant-eating insects will respond to pheromone calls, too.

For Darwin, the most spectacular of all plant behaviors were produced by carnivorous species that trap animals. Darwin called the behavior of the Venus flytrap "one of the most wonderful in the world" (Ellison and Gotelli 2009). Insects that crawl across the pad of a flytrap brush over tiny hairs, which release electrical signals to other cells in the plant (FIGURE 13.6). They trigger a series of molecular events inside the pads that cause them to snap shut in less than a second. The plant then releases enzymes to digest its prey.

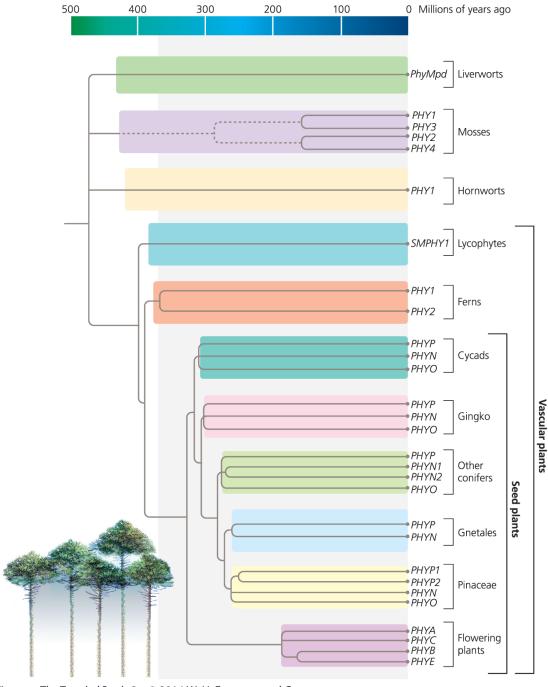


Marty Pitcairn/Shutterstock.

Plants have their own types of behavior. Venus flytraps sense insects and respond by snapping their pads shut and then slowly digesting their prey.

The study of the evolution of plant behavior is a flourishing area of research. Consider the responses plants have to shade. Those responses depend on light-sensing pigments called phytochromes. All plants have cytochromes, suggesting that they were present in the earliest plants that moved on land over 430 million years ago. But starting about 360 million years ago, many plant lineages underwent a burst of duplication of phytochrome genes (FIGURE 13.7). This explosion coincided with the first forests on land, where plants formed a canopy that cast the ground in dark

shade. The new phytochromes evolved to be sensitive to different wavelengths of light, expanding the behaviors that plants could carry out in response to changes in their environment (<u>Mathews 2006</u>).



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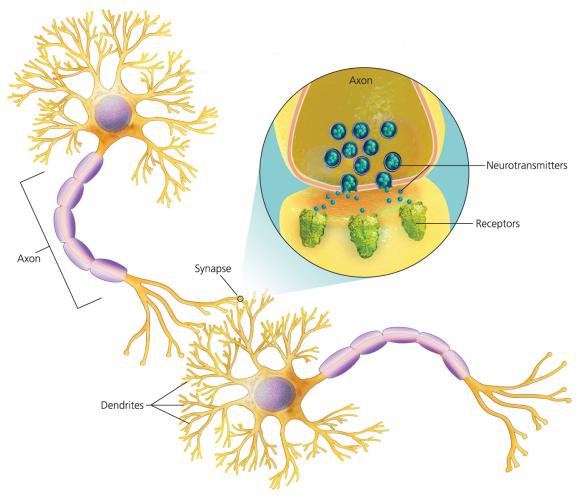
Plant behavior has evolved much like animal behavior has. Plants adjust the direction and speed of their growth in response to changing levels of light. The genes for their light-sensing pigments, called phytochromes, underwent several rounds of duplication over the past 360 million years. The onset of these duplications coincided with the emergence of the first forest canopies, which shaded the plants on the forest floor. (Information from Matthews 2006.)

The Origin of Nerves

Animal behavior is unusual compared to other clades because it emerges from a unique type of cell: the neuron. A neuron generates signals with pulses of electric charge that move from one end of the cell to the other. They can jump to a neighboring neuron at a special junction called a synapse, into which the signal-carrying neuron dumps chemicals. The neighboring neuron takes the chemicals up, triggering it to send a signal of its own. If you see a basketball coming toward you, the sensation travels from your eye through the optic nerve to the brain, where signals move through a dense network of some 86 billion neurons. Your brain then sends out commands to your body so that you can catch the ball.

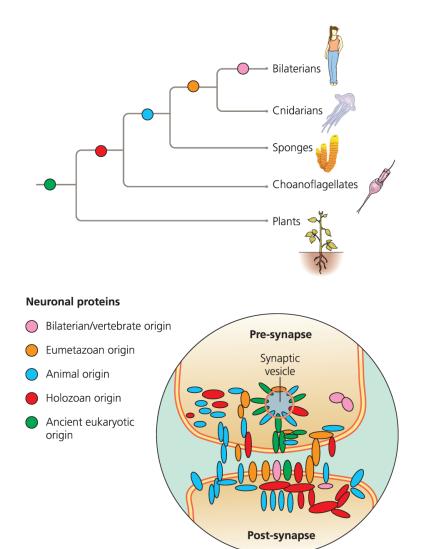
Animal nervous systems vary tremendously. Jellyfish have no centralized brain; instead, they grow a diffuse ring-shaped net of neurons that spread across their whole body. The nematode worm *Caenorhabditis elegans* has only 302 neurons in its entire body. But along with this variation, animal nervous systems share many homologies that are the result of their common descent. All neurons form synapses, for example; the neurotransmitters that influence their activity are for the most part identical.

Yet the animal nervous system did not evolve in a single step. Instead, its components gradually emerged over hundreds of millions of years. The synapse, for example, depends on a large network of proteins to carry a signal from one neuron to another (FIGURE 13.8). Many of the genes for those proteins are also present in eukaryotes outside the animal kingdom—even in plants (FIGURE 13.9). This pattern suggests that ancient genes involved in other functions in cells were recruited in animals to form the synapse (Srivastava et al. 2010).



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All animals (except for sponges) use neurons to control their behavior. Spikes of voltage travel down the length of a neuron and then trigger the release of neurotransmitters at synapses, which in turn trigger activity in neighboring neurons.



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The network of proteins in neurons evolved in a stepwise fashion long before neurons existed. Some of the proteins in the synapse are colored in this diagram to indicate when they evolved. (Information from <u>Srivastava et al. 2010</u>.)

To understand the origin of neurons, it's especially useful to study sponges. While they may not look like animals, they very much are. Their cells share many features found only in animals, such as special glue-like proteins that hold their cells together. Yet they have no nervous system, nor any cell that clearly corresponds to a neuron. Both the fossil record and studies on DNA indicate that sponges split off from the ancestors of all other living animals

perhaps as long ago as 800 million years. The origin of a full-blown nervous system must have occurred after that split.

Yet sponges have a type of cell that some scientists have dubbed a "protoneuron" (Jékely 2011). When sponges reproduce, their fertilized eggs develop into tiny larvae that swim through the water until they settle back to the ocean floor (FIGURE 13.10). These grape-shaped larvae have a crown of hairs at one end, which beat back and forth to propel the sponge. Scientists have found that these hairs contain proteins that are homologs to some of the proteins found in true neurons. It's possible that these hairs combine the two functions of neurons: sensing the environment and responding with a behavior. Such a dual-action cell might have given rise to the specialized neurons found in other animals, whose nervous systems include some neurons that only deliver sensory information and other neurons that only send out commands.

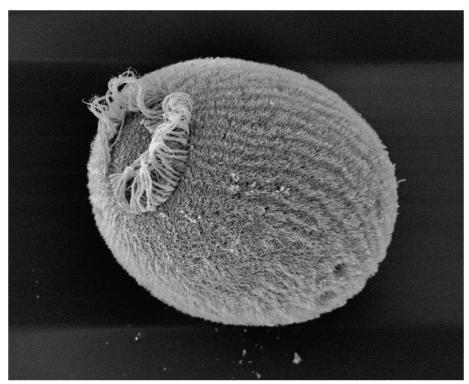


Photo by Sally Leys.

FIGURE 13.10

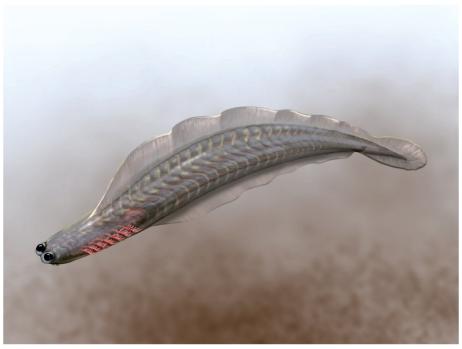
Sponges lack neurons, but their larvae have neuron-like hairs (the ring in this picture). These hairs express genes found in true neurons, and they appear to sense the

environment and respond by changing their beating pattern. It's possible that neurons evolved from such cells.

The Vertebrate Brain Is Not an All-Purpose Computer

The earliest nervous system that evolved in the ancestors of bilaterians and cnidarians was probably a diffuse net. Only after the split between bilaterians and cnidarians did our ancestors evolve a central nervous system, organized around a brain. In our own vertebrate lineage, the brain evolved to be especially large and complex.

The oldest signs of the vertebrate nervous system can be found in 530-million-year-old rocks in China. Those rocks contain hundreds of fossilized impressions of a tiny creature called *Haikouichthys* (Shu et al. 2003). Measuring about 3 centimeters long, it has many (but not all) of the hallmarks of living vertebrates (FIGURE 13.11). Its spinal canal is surrounded by vertebrae, which are supported by a notochord. It has a series of pouches and arches to support gills. Two dark spots at the front of its body appear to be simple eyes. It has holes on the side of its head where sound-sensitive nerves probably grew, and it has another cavity up front that paleontologists suspect was a nostril for smelling. Its head contains a mass of cartilage that appears to have surrounded a primitive brain.



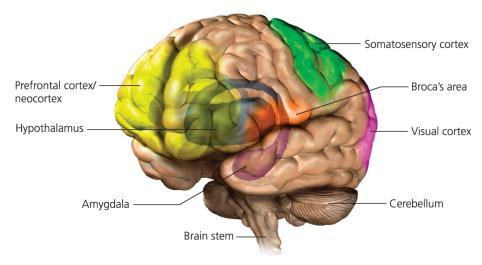
Carl Buell.

A key step in the evolution of vertebrate behavior was the evolution of a brain. The earliest fossil evidence of a brain is from the 530-million-year-old *Haikouichthys*.

Over the next 100 million years, fish lineages evolved from such humble creatures into the biggest animals in the world. They became predators that could search for prey, in many cases chasing other animals down. Many of the major features of our nervous systems evolved during this transition. As fishes evolved longer bodies, their neurons evolved to great lengths as well. This transformation could not have occurred without the evolution of myelin. This tissue forms an oily sleeve around neurons that acts like the insulation around a wire, preventing the loss of electrical signals over long distances. These lengthened neurons began to supply the vertebrate brain with information from larger sense organs, and new motor neurons allowed fishes to steer their bodies in complex ways.

We can learn a great deal about the organization and function of the human brain (FIGURE 13.12) by exploring its evolutionary history. In other vertebrates, we can see homologous regions of the brain, such as the cerebellum, the optic tectum, and the cerebrum (FIGURE 13.13). Many of these

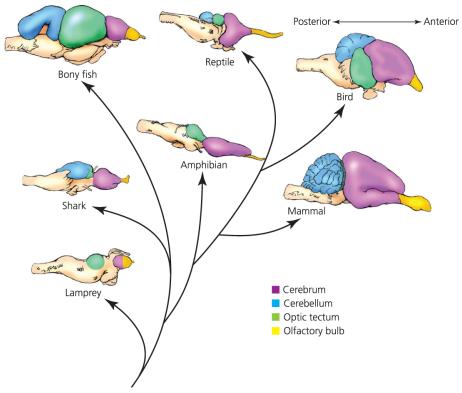
regions continue to perform functions they performed hundreds of millions of years ago. The cerebellum, located at the base of the brain, is especially important for balance, for example. People who suffer damage to their cerebellum have trouble walking. Fish use their cerebellum to stay balanced in water.



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FIGURE 13.12

The human brain, like other vertebrate brains, is divided into many specialized regions, each helping to carry out certain functions. The cerebellum, for example, is important for balance. The somatosensory cortex organizes sensory information from the skin. Broca's area is involved, among other things, in language. Neuroscientists can find homologs for many of these areas in other animals.



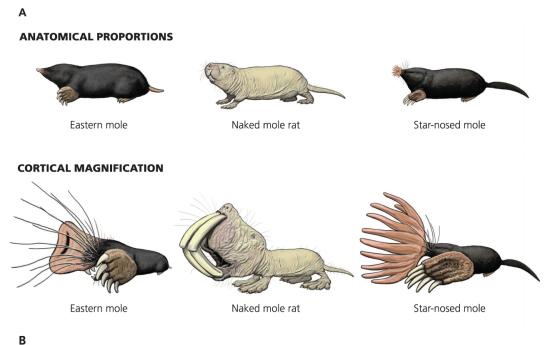
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Vertebrate brains come in many shapes and sizes. Despite these differences, they share the same basic organization. (Information from <u>Kardong 2002</u>.)

As the vertebrate lineages diverged, their brains diverged as well. In some sharks, the cerebellum is larger than the other sections, while in salmon the optic tectum is larger. Larger cerebrums tended to evolve in the vertebrates that moved onto land—the tetrapods. In mammals, the outer layer of the cerebrum, known as the cerebral cortex, expanded drastically. In humans, the cerebral cortex now takes up 90 percent of the brain.

The cerebral cortex is especially important for our most sophisticated kinds of thinking—for recalling memories, for using language, for making tough choices. As it develops, the cerebral cortex becomes parceled into some 200 distinct areas. The neurons in any particular area are densely interconnected, and together they help with certain mental tasks. One area, called the somatosensory cortex (FIGURE 13.14), receives signals from touch-

sensitive neurons in the skin. Another area, the visual cortex, maps the signals from the eyes and then sends them to other areas of the brain.





Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Bottom photo: Sensory Homunculus (plaster)/English School, (20thcentury)/NATURAL HISTORY MUSEUM, LONDON /Natural History Museum, London, UK/Bridgeman Images.

FIGURE 13.14

The brains of mammals are adapted to the ecological niches in which they live. For example, each mammal has some regions of the body that are very sensitive, and

others that are not. A: These images show the relative concentration of neurons dedicated to each part of the body in the somatosensory cortex of the brain. *Left:* Moles use their forelegs to burrow, and their noses and whiskers to sense prey. These regions are the biggest in their body map. *Middle:* The naked mole rat, which spends its life digging tunnels through dry earth, has a body map dominated by its teeth, mouth, and feet. *Right:* Star-nosed moles, on the other hand, have fleshy appendages on their noses, and these "stars" dominate their body maps. B: This sculpture represents the human body map in the somatosensory cortex. Unlike rodents, our hands and mouths are most sensitive—reflecting our adaptations for using tools and language. (Rodents information from Alcock 2004.)

As vertebrates adapted to different ecological niches, their brains evolved to become specialized for certain kinds of information. Neuroscientists map the somatosensory cortex of animals by touching parts of their skin and recording the responses of neurons in the brain. Some parts of the body are better represented in this body map than others. Neuroscientists chart these patterns by making drawings of an animal's body, making highly sensitive parts bigger and those with fewer sensory inputs smaller. These sensory maps can differ dramatically for species with different ways of life.

The sensory maps of some burrowing animals are shown in Figure 13.14A. An eastern mole has extremely sensitive front feet, nose, and whiskers. Those are the parts of the body that it uses to dig through the dirt and to sense the presence of insects that it can eat. A star-nosed mole, by contrast, has long, fingerlike extensions around its nose that it uses to probe the mud around streams. That tiny patch of skin is more sensitive than the rest of its body. Naked mole rats also burrow through the ground, but they live in arid regions of Africa, where they use their teeth to dig tunnels. For them, the teeth rather than the nose dominate their somatosensory cortex.

We humans are also biased about the information we receive, but in a different way. As <u>Figure 13.14B</u> shows, our hands, lips, and tongues are strongly represented in the somatosensory cortex. This pattern is a result of our own ecology—particularly our adaptations for using tools with our hands (see <u>Chapter 14</u>).

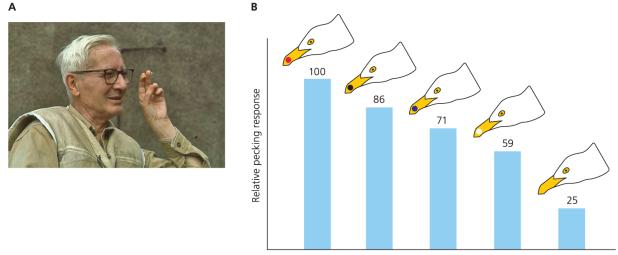
Born to Peck

Our behavior emerges from the biology of our nervous system: from the ways in which signals are processed by our network of neurons that have evolved over the past 800 million years. Many behaviors in humans and other animals have evolved as quick, automatic responses to our environment.

The study of these responses was launched by the Nobel Prize—winning expert on animal behavior, Niko Tinbergen (Kruuk 2003). In one of his most famous studies, Tinbergen studied herring gull chicks. He had observed that when the adult gulls approach their nest, their chicks beg and peck at their bills. The adult gulls respond by regurgitating their prey, which the chicks feed on.

It's a remarkably reliable response, one that the gulls are born with. Tinbergen wondered what information the chicks used to generate it (<u>Tinbergen and Perdeck 1950</u>). Were they responding to the whole body of the parent bird, he wondered, or just to some feature of it? To find out, Tinbergen ran an experiment in which he showed the chicks cutouts of gull heads. In one case, he showed the chicks a detailed cutout of an adult bird head, complete with the distinctive red spot that herring gulls have on their beak. In another, he showed the chicks a cutout head without the red spot. In still other cases, he showed them heads with dots of other colors, including black, blue, and white.

The red-colored dot proved to be the most important feature for triggering the begging in gull chicks (FIGURE 13.15). In other words, the chicks did not need to do any sophisticated three-dimensional visual processing in order to obtain food. A bias in their sensory systems, making them highly sensitive to a red dot, was sufficient.



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A: Niko Tinbergen ran groundbreaking experiments that revealed the nature of innate behaviors in animals. Herring gull chicks respond to the sight of their parents by begging for food. To determine what the chicks respond to, Tinbergen built cardboard cutouts of adult gull heads. A cutout of the beak alone is almost as effective at triggering a response from a chick as a full head. B: On the other hand, a head without a red spot is less likely to trigger a pecking response. These experiments show that the red beak spot is mainly responsible for a chick's response. (Information from <u>Tinbergen and Perdeck 1950</u>.)

Gull chicks peck in response to a red dot the very first time they see one. This reliable response does not mean there's a special gene "for" bill pecking in the gull genome. The response emerges as the bird's nervous system develops, as its eyes begin to recognize shapes and colors, and as it gains control of the muscles in its neck and head. Nevertheless, gulls reared in a normal environment will almost always begin to peck at bill-shaped objects with red dots the first time they see them. The many genes that work together to produce this behavior are passed down from gulls to their chicks.

The genes that encode these innate behaviors evolve just like other genes. Stevan Arnold, a biologist at Oregon State University, has documented the evolution of behaviors in terrestrial garter snakes. These snakes, which live in the western United States, generally feed on animals like fish and frogs. In some areas near the California coast, however, they eat slugs that are

abundant in the damp coastal forests. To see how the snakes had acquired these tastes, Arnold collected female garter snakes carrying embryos and brought them to his lab.

When the baby garter snakes were born, Arnold began to offer them food. The coastal snakes refused chunks of fish, but they eagerly feasted on pieces of slug (Arnold 1981a). Even a cotton swab that smelled of slugs caused them to attack. Most inland snakes refused the slugs, and many refused to eat them until they were on the verge of starvation. To test whether these populations had diverged genetically in their preference for slugs, Arnold interbred the inland and coastal snakes in his lab. Their hybrid offspring showed more interest in slugs than their inland parents did, but less than their coastal parents did (Arnold 1981b).

Arnold's results demonstrated that different behaviors can evolve in populations of the same species, and it's likely that this evolution happens rapidly. Additional studies on the DNA of terrestrial garter snakes show that they were restricted to a few refuges during the last ice age and rapidly moved into new territories as the glaciers retreated. Inland and coastal populations shared a common ancestor at that time, so their different foraging preferences must have evolved within the last 12,000 years.

Rewiring Behavior

While animals reliably develop some behaviors, others they adjust by learning. Consider a simple behavior experiment in which a rat learns that if it presses a lever in response to a flashing light, it will get a piece of food. The rat is forming a connection between networks of vision-processing neurons and neurons involved in planning movements. This connection is formed through changes in the synapses that allow neurons to communicate with one another. Some synapses become stronger and others weaker. New synapses join together neurons that were not previously linked.

Learning is essential for the reproductive success of many animals, allowing them to adapt to changing circumstances. They can learn how to find food more efficiently, for example, and come to recognize new threats. Fear itself, it turns out, can be learned. Psychologists can train people to have new fears by having them listen to a series of sounds. Most of the sounds are just ordinary tones; but from time to time, the psychologists play a loud, painful sound that triggers an anxious response from the subjects (responses can be measured with electrodes on the skin). If the psychologists consistently play a particular note—say, middle C—before the painful sound, their subjects will unconsciously learn that middle C means trouble ahead. Just hearing middle C will trigger a fear response, even if no painful sound follows.

Individuals vary in their ability to learn, and some of this variation is genetically based. As a result, learning itself can evolve. By running experiments on fruit flies, Tadeusz Kawecki and his colleagues at the University of Fribourg in Switzerland have observed as learning evolves in their laboratory (<u>Burger et al. 2008</u>; <u>Mery and Kawecki 2003</u>).

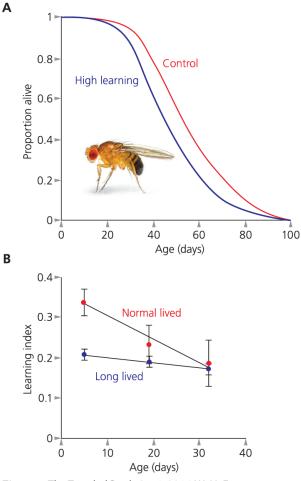
Kawecki and his colleagues offered the insects a choice of orange or pineapple jelly to eat. One of the dishes of jelly also contained bitter-tasting quinine. Within a few hours, the flies developed a strong preference for the quinine-free jelly. They learned to associate the sweet smell of the other jelly with the nasty surprise in store if they had a taste. It didn't matter whether

Kawecki spiked the orange jelly or the pineapple jelly with quinine—in either case, the flies learned to avoid the noxious flavor.

Some of the flies learned to associate the quinine with a particular flavor of jelly faster than others, and Kawecki and his colleagues discovered that fast-learning flies tended to produce fast-learning offspring. They set up an experiment to select for this trait. They gave flies three hours to learn which jelly was laced with quinine, after which the flies mated and then laid eggs on either flavor of jelly. Kawecki and his colleagues collected the eggs from the quinine-free dish and reared them for the next generation. Some of the eggs came from flies that had laid their eggs on the dish by chance; others had managed to learn that the smell of a particular flavor of jelly meant that the jelly tasted bad.

The second generation now faced the same challenge, except that the scientists had switched the quinine to the other flavor of jelly. In the third generation, they switched back. Kawecki and his colleagues predicted that this procedure would foster the evolution of general learning, rather than an instinctive attraction to one particular flavor. All told, they reared the flies through 15 generations of selection. They then compared these flies with control lines that had been reared for 15 generations without selection. They found that the selected lineage needed less than an hour to learn to avoid a quinine-laced dish of jelly. Flies from unselected control lines, on the other hand, needed several hours. A population of fast-learning flies had evolved in just generations.

The "smart" flies pay a price for fast learning, however, as the scientists discovered when they mixed larvae of smart flies with ordinary ones. The smart flies had shorter life spans than the control flies (FIGURE 13.16). The scientists got a similar result when they ran an experiment in which they selected flies for longevity. Kawecki and his colleagues found that longevity-selected flies were slower at learning than wild-type flies.



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Photo: Roblan/Shutterstock.

FIGURE 13.16

A: Tadeusz Kawecki and his colleagues at the University of Fribourg in Switzerland ran an experiment in which they selected for flies that could learn quickly. These flies died sooner than controls. B: Flies selected for longevity, on the other hand, turned out to be poor learners. (Data from Burger et al. 2008.)

Kawecki's experiments demonstrate that the evolution of learning, like the evolution of other aspects of behavior, may be constrained by trade-offs. Learning imposes some kind of cost on an organism. Forming a lot of connections between neurons may cause harmful side effects, possibly by producing cell-damaging molecules. It's also possible that the proteins used

by neurons during learning interfere with other chemical reactions, such as the ones that repair cells.

Natural selection can favor increases in learning only if the costs are outweighed by their benefits. That balance is different for each species. Learning may be favored when a species cannot rely on innate responses—that is, when its environment becomes less predictable. Learning may raise the fitness of bees that feed on many different species of plants, each with a differently shaped flower and a different flowering time. For bees that feed on only a single type of flower, learning may not provide enough benefit to outweigh its cost. Kawecki's experiments demonstrate that flies have the genetic potential to become better learners, but only under his lab conditions did evolution actually move in that direction. In the wild, this kind of change may impose too high a cost.

Shortsighted Selection and Selfishness

In the past few examples of the evolution of behavior we've considered—such as snakes and flies recognizing good-tasting food—we've focused only on the behavior of individual animals in isolation. Yet in many cases, animals do not live in isolation. They live in swarms, hives, and herds. Much of their behavior is directed to other members of their group—baboons grooming one another, young male wolves challenging older ones for dominance, thousands of sardines traveling together in a single school. One of the biggest questions in the evolution of behavior is why so many species are social.

The debate over social behavior exploded in the mid-1900s, as scientists were beginning to gather the first detailed, long-term observations on animals. Some researchers argued that selection acts almost entirely on the behavior of individuals, while others maintained that a significant amount of behavior was the result of selection acting on entire groups.

Individual selection occurs when alleles cause particular individuals within a population to perform better than other individuals, causing the alleles to spread. When males compete over access to females, for example, the successful males can transmit more copies of their alleles to subsequent generations. But some biologists argued that selection could act on groups as well. Some groups might survive better than others. The alleles of the animals in that group would be favored by selection.

The biologist V. C. Wynne-Edwards argued that this form of group selection was an important factor in the evolution of behavior (<u>Wynne-Edwards 1962</u>). If the animals in a group ate food at a limited rate, they could avoid destroying their food supply and therefore starving. Wynne-Edwards and other group selection advocates argued that such groups would survive while less cooperative groups would perish.

The British biologist John Maynard Smith and the American biologist George Williams pointed out a problem with Wynne-Edwards's group selection argument: populations of self-sacrificing individuals are not evolutionarily stable (<u>Maynard Smith 1964</u>; <u>Williams 1966</u>). If individuals acquire mutations that make them behave selfishly, they will take over a group of cooperators. The selfish individuals will have higher fitness relative to the cooperators, and their genotypes will replace the cooperative genotypes in the population. As we saw earlier in this chapter, Kessin and his colleagues saw precisely this transformation take place in their studies on cheating slime molds (<u>Dao et al. 2000</u>).

Scientists who study the evolution of behavior now generally agree that—except for a few unusual circumstances—selection operates at the level of individuals and not at the level of group versus group (but see Wilson and Sober, 1994, for a debate on this issue). Selection on individuals is typically much stronger than selection acting on groups. The behaviors that we observe in natural populations today are there because individuals performing those actions did better—they were more likely to survive, and more successful at reproducing—than individuals that behaved in other ways.

Why Be Social?

If individual selection can undermine group selection so easily, it raises an important question: why are there any stable groups at all? Evolutionary biologists have found that individual selection can indeed favor group living if the gains it brings to an individual outweigh the costs (FIGURE 13.17).



(A) cascoly/Getty Images; (B) age fotostock/age fotostock; (C) MartinMaritz/ Shutterstock.

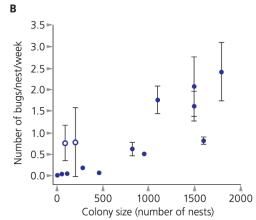
Many animals form groups to reduce their chances of being attacked. A: By jumping off of ice shelves together, Adélie penguins reduce their chances of being eaten by sea lions waiting in the water. B: Fish avoid being eaten by dolphins and other predators by forming schools. C: Ostrich chicks are less likely to be captured by predators if they surround themselves with other chicks.

Individuals can sometimes gain more protection from predators in a group than on their own, for example. A single antelope cannot spend all its time on the lookout for leopards, or it will starve. In a herd of antelopes, on the other hand, individuals can spend much of their time eating because there are always at least a few antelopes looking around for a predator. And even if a predator does strike a group of antelopes, each group member faces a smaller risk.

Adélie penguins, which live on the coast of Antarctica, vividly illustrate how group living allows individuals to escape predators. They dive office sheets into the ocean to search for fish; but when they take the plunge, they sometimes get eaten by leopard seals cruising just offshore. Rather than jump in alone, the penguins crowd together by the hundreds and then leap en masse. Together, they can overwhelm the leopard seals, which can't focus on any single individual. The huge numbers of jumping penguins may also create a dilution effect because the likelihood of any individual being caught is reduced.

While living in groups can bring many benefits, it can impose costs as well. Individuals living in big groups are at greater risk of getting sick, for instance. Parasites and pathogens can spread more effectively in dense groups of animals than among solitary animals (FIGURE 13.18). By measuring the costs and benefits of group living in particular cases, biologists can explain why individual members of social groups behave in the ways they do.





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A: Cliff swallows build nests in dense colonies. B: This group living comes at a cost: in large colonies, each nest becomes infested with more parasites. (Information from Brown 2004.)

The Importance of Kin

In many animal groups, individuals don't just help each other catch prey or escape from predators. They also help each other raise their young. During the debates over selection for behavior, such altruistic behaviors seemed to be powerful evidence in favor of group selection. Helping other members of the group benefited the group as a whole. Altruism seemed to pose a serious problem for individual selection because shortsighted natural selection seemed incapable of favoring individuals who lowered their own fitness—in terms of the time spent foraging or rearing young—for the benefit of other individuals.

Since the 1960s, however, a great many studies have documented a number of situations in which helping raises fitness when compared to other behaviors. Consider, for example, a young meerkat (FIGURE 13.19). If it lives on its own, it faces a higher risk of being killed by a predator than if it stays with a group. The cost of living in the group is having to help rear the offspring of the dominant meerkats. That may be a small price to pay for group membership if it improves the helper's odds of surviving to another season.



namibelephant/Getty Images.

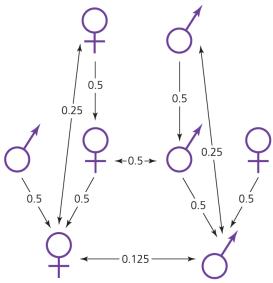
FIGURE 13.19

Meerkats live in large groups in southern Africa. The subordinates in a group may spend years helping the dominant members rear their young rather than having offspring of their own. This behavior helps increase the size of the entire group, which directly raises the fitness of the subordinates.

Helping may also allow individuals to gain valuable experience that will serve them well when they eventually breed. It may also give them opportunities to acquaint themselves with future mates. In some species of birds, helper males assist other males by bringing food for their mates and their young. If the male getting the help dies, the helper male has a better chance of mating with the female (<u>Clutton-Brock 2002</u>). Helping may even be the best way to acquire a nesting territory, if it can be inherited from the breeders when they die.

In the 1960s, the British biologist William Hamilton offered a new way of thinking about cooperative behavior. He noted that an animal shares 50 percent of its alleles with one of its offspring, but it also shares 25 percent of its alleles with its nephews and nieces (FIGURE 13.20). If an animal helps its sibling to reproduce, some of its own genes will be carried down to the next generation. Helping relatives has an evolutionary cost, because an animal

reduces its own reproductive success; but that animal may also be able to spread some of its own alleles by helping its relatives.



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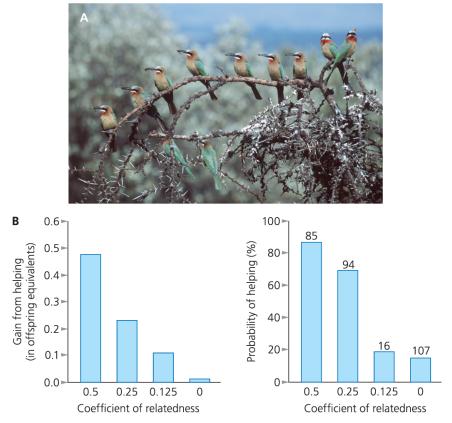
FIGURE 13.20

In diploid organisms (with two copies of each chromosome), each parent transmits 50 percent of its genetic information to each offspring. Here, the first generation of parents are shown in the top row, and their offspring are in the second row. On average, siblings share half of each other's parent's contributions to their genome. Scientists express this sharing of DNA with the "coefficient of relatedness." Zero means no shared DNA, and 1 means that two individuals have identical DNA. Two siblings will, on average, have a coefficient of relatedness of 0.5. The bottom row are two members of the third generation. They have a coefficient of relatedness of only 0.125, and they have a coefficient of 0.25 with their grandparents. The fact that relatives share some of the same alleles fosters the evolution of altruism toward kin. (Information from Brembs 2001.)

Hamilton argued that the original definition of fitness—based on how many offspring are produced by organisms with a particular genotype—should be updated. The definition also needed to include the effect an individual had on the reproductive success of its relatives, which carry the genotype as well. Hamilton proposed that this new kind of fitness be called

inclusive fitness. He argued that altruism evolved if the cost of helping others was paid back in a rise in inclusive fitness.

In the years since he presented this argument, evolutionary biologists have developed much more nuanced mathematical models and have run experiments to test their predictions. Hamilton's rule sheds light on many behaviors in animal groups. It explains, for example, why African birds called white-fronted bee-eaters sometimes help other birds rear their young: they can ensure that more copies of their genes are replicated than if they were to try to reproduce themselves. As Hamilton would have predicted, the chances of their helping another bird are greatest for close relatives and lowest for unrelated bee-eaters (FIGURE 13.21).



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FIGURE 13.21

A: Instead of attempting to breed in their first two years of life, white-fronted bee-eaters often help other members of their colony raise their own young. Cooperative behavior in this species occurs most frequently during harsh years, when the chicks of young adults

would be likely to die. B: The most help that the birds get comes mainly from close relatives. Likewise, a bird is most likely to help rear offspring that are closely related to it. (Data from <u>Emlen and Wrege 1988</u>, <u>1989</u>.)

Green Beards

For an organism to raise its inclusive fitness, it needs a way to distinguish kin from unrelated individuals. In many species, animals learn to recognize a physical feature of their relatives before they leave the nest; they may also recognize a distinctive odor. Sometimes they can even use odor cues to recognize relatives they have never encountered before.

Hamilton suggested that kin recognition might evolve thanks to an allele that causes its bearer to display a distinctive trait and also causes the animal to behave altruistically toward the individuals that display it. Richard Dawkins nicknamed such a trait a "green beard" (<u>Dawkins 1976</u>). It was plausible, but for nearly 30 years after Dawkins described it, scientists weren't sure how such an effect might actually work. It seemed to demand too many functions from a single allele. In 2003, however, David Queller, Joan Strassmann, and colleagues (then at Rice University) discovered an allele with green beard effects in the cellular slime mold *Dictyostelium discoideum* (<u>Queller et al. 2003</u>).

When slime mold cells begin to coalesce and form slugs, they preferentially aggregate with relatives. This preference is probably adaptive because it means that when some of the cells die in order to make a stalk, they are sacrificing their lives for relatives rather than for slime molds that do not share many alleles with them.

Queller and Strassmann discovered that *Dictyostelium* recognize their kin thanks to a gene called csA. It encodes a protein that forms on the surface of a *Dictyostelium* cell. Its structure allows it to hook onto the same protein on other cells. As slime mold cells aggregate to form a slug, cells with the same csA protein stick together. Slugs with knockout mutations to the *csA* gene get left behind. They do not have the appropriate allele, and so they can't benefit from the sacrifice of the stalk-forming cells.

In many vertebrates, a family of immune system genes plays an important role in kin recognition. Genes in the major histocompatibility complex (MHC) code for proteins that recognize and grab fragments of pathogens or parasites inside cells and shuttle them to the cell surface, where they can be

attacked by T cells of the immune system. Because MHC genes must defend against a great diversity of microbes in the environment, the MHC genes themselves must be diverse. And they are stunningly so. For each of the nine principal MHC genes in humans, there are between 800 and 1600 different alleles. As a result, the combinations of alleles vary greatly among individuals. They also serve as valuable markers for recognizing relatives.

Wayne Potts, a biologist at the University of Utah, and his colleagues have found that mice can recognize individuals with the same alleles at their MHC loci (Penn and Potts 1998). House mice form communal nests and nurse each other's pups indiscriminately. Not surprisingly, females benefit if they can preferentially team up with relatives so they derive inclusive fitness benefits from communal nesting. One of the ways they do this is by recognizing allelic similarity at MHC loci: mice are more likely to nest communally with partners who share allelic forms of their MHC genes.

Animal Geniuses

While most animals can learn, some species—like the New Caledonian crows—have taken learning to extraordinary extremes. When we watch these crows make a tool to get another tool, it is tempting to refer to them as "smart." We have to be very careful with the words we choose, however, because anthropomorphism can cloud our understanding of animal behavior. Nevertheless, it is unquestionably true that some species of animals are capable of complex cognition.

Complex cognition takes many forms in the animal kingdom. In some cases, it allows animals to have sophisticated social interactions with other members of their species. Bottlenose dolphins, for example, can recognize individual members of their group by their distinctive whistles (<u>Janik</u>, <u>Sayigh</u>, <u>and Wells 2006</u>). Vervet monkeys produce certain kinds of screams for certain kinds of predators (<u>Seyfarth</u>, <u>Cheney</u>, <u>and Marler 1980</u>). This repertoire of alarm calls is adaptive because it helps neighboring monkeys—who typically are close relatives—to escape. If an eagle is swooping down at them, they'll want to scramble out of the trees; but if a leopard is running toward them, a tree is exactly where they want to be.

Chimpanzees, our closest living relatives, make an even wider range of sounds, and they can also communicate with gestures—something not observed in other nonhuman primates (FIGURE 13.22). They may wave their arms, reach out their hands, or slap the ground. Chimp gestures generally convey some kind of request—to play, for example, or to share some food—but there's no deep biological impulse linking one gesture to one meaning. In fact, much like human cultural rituals, chimpanzee gestures vary from population to population (Pollick and de Waal 2007).



Mary Beth Angelo/Science Source.

FIGURE 13.22

Chimpanzees use many different gestures to communicate with each other. Two populations of chimpanzees may give two different meanings to the same gesture. Some researchers have suggested that gestures might have played a critical part in the evolution of human language.

Chimpanzees can also adjust their sounds depending on who's listening. When wild chimpanzees are attacked by other members of their group, for example, they give a distinctive scream. Sometimes, bystander chimps will respond by intervening and breaking up the fight. Katie Slocombe and Klaus Zuberbühler, two primatologists at the University of St. Andrews in Scotland, have observed that when attacks were more severe, the victims made higher-pitched screams that lasted longer and came in longer bouts. But when highranking chimpanzees were nearby, the victims would scream as if the attacks were much worse than they really were. Slocombe and Zuberbühler suggest that the victims are trying to take advantage of the power of high-ranking chimpanzees in their area (Slocombe and Zuberbühler 2007).

Despite these sophisticated forms of communication, chimpanzees can fail some seemingly simple cognition tests. One of them is known as the pointing test. A scientist presents a chimpanzee with two cups, one of which has a piece of food under it. The scientist points at the cup with the food, and the chimpanzee then gets to choose one of them. Chimpanzees are equally likely to pick either cup. In other words, they don't pay attention to the pointing cue. It turns out that two species can spontaneously pass the pointing test: humans

and dogs. Even a puppy will tend to pick a cup that a human points at (<u>Hare and Tomasello 2005</u>, <u>Kaminski et al. 2009</u>). Dogs excel in this particular social task thanks to their intense selection for social interactions with humans. The tame Siberian foxes turn out to do as well at the pointing test as dogs.

Complex cognition can also allow animals to manipulate their surroundings in sophisticated ways—most impressively by making and using tools, as we saw with New Caledonian crows. Chimpanzees also use tools, and over the past 50 years scientists have catalogued a long list of different chimpanzee tools. They crack nuts by placing them on one rock and then smashing them with another, for example. They use a stiff digging stick to punch a hole into a termite nest and then use a slender, flexible one to fish out the insects. Chimpanzees also fashion sharp spears to stab bush babies (McGrew 2010).

Animals make tools not just to get food, but to protect themselves. Gorillas will poke a stick into water to test its depth. Orangutans will fashion twigs into probes they can use to pull out irritating hairs from the fruits they eat. Dolphins in Shark Bay, Australia, stick sponges on their rostrums to protect their sensitive skin as they probe the rough floor of the bay for food (FIGURE 13.23; Seed and Byrne 2010).



Ewa Krzyszczk/Shark Bay Dolphin Project.

A dolphin in Shark Bay, Australia, wears a sponge to protect its rostrum (its long mouth) as it hunts for prey on the rocky seafloor. Dolphins are among the few lineages of animals known to make and use tools.

When behavioral ecologists encounter an example of complex cognition in animals, they consider both its proximate and ultimate causes. This approach was pioneered by Tinbergen, who recognized that the ability to make a tool and use it requires more than just innate habits, such as pecking at a red dot. It demands that an animal recognize its long-term goal and find a way to reach it. New Caledonian crows, for example, don't simply start prying at sticks at the sight of food. In Taylor's experiment, the birds recognized a long-term goal—getting food out of a box—and then recognized that a short stick would be insufficient for the job. Reaching a goal can also demand the ability to plan ahead. New Caledonian crows transport their tools long distances before using them.

Toolmaking also demands a capacity for innovations—for coming up with new solutions that other animals haven't discovered before. New Caledonian crows can bend pieces of wire to make larva-fishing hooks, for example. Innovations require not just the ability to picture a goal, but an understanding of physics—how different objects will function in response to gravity, friction, and other forces.

These studies help uncover the proximate mechanisms of tool use in animals. But to understand the ultimate mechanisms behind tool use, scientists reconstruct its evolution. One method is phylogenetic. Scientists examine the pattern of the emergence of a particular behavior (see Chapter 4 for a discussion of mapping the evolution of traits on a phylogeny). Toolusing animals are limited to amniotic lineages—birds and mammals. But within the amniotes, tool use has arisen independently on several occasions. We know this because toolmaking animals—dolphins, primates, and crows—do not share a close common ancestor. Primates are much more closely related to shrews than to dolphins, for example, and no shrew has ever shown the slightest ability to make a tool.

Such patterns raise interesting questions. Is there something about the amniotic nervous system that makes amniotes especially prone to evolving tool use? And did dolphins, primates, and crows all experience similar

environmental conditions that drove their evolution into full-blown tool users? Why didn't more animals evolve into tool users?

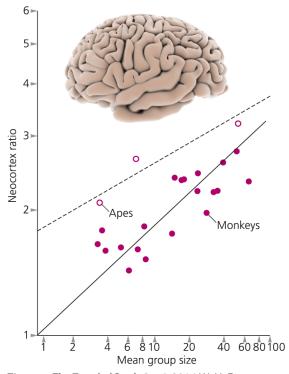
Using tools demands time and effort. While a tool-using animal is busy struggling with its equipment, other animals may be able to get food with nothing but their mouths. When food is scarce or unpredictable, the costs of making tools may be outweighed by their benefits. Capuchin monkeys that live in dry forests in Brazil will use stones to dig for tubers, for example, while the ones that live in moist forests with abundant fruits have never been observed digging tubers (Seed and Byrne 2010).

Christian Rutz of the University of Oxford and his colleagues tested the hypothesis that scarcity fosters the evolution of toolmaking by examining the diet of New Caledonian crows (Rutz et al. 2010). First they measured carbon and nitrogen isotopes in the feathers and blood of the birds, and then they compared the results to those from the foods the birds ate. Rutz and his colleagues found that a substantial amount of the crows' protein and lipids came from the insect larvae that they obtained with tools. The great benefit of food that can be obtained only with tools could have driven the extraordinary behavior of the New Caledonian crows.

Another hypothesis worth considering is that tool use evolves only in lineages that have already acquired skills such as following goals, planning, and understanding rules. New Caledonian crows are closely related to other species of crows, as well as jays, in the Corvidae family. Other corvids cannot match New Caledonian crows in using tools, but they are capable of other kinds of complex cognition. Scrub jays, for example, can hide seeds in thousands of caches over many square miles, and they can accurately recall their location. Once a lineage evolves this level of cognition, toolmaking may become a possibility.

Another possible prerequisite for tool use is complex social cognition. At first, this might not seem to make much sense. When we think of social cognition, we might think of organizing a food drive or starting a company. And when we think of tool use, we think of sitting alone in front of a computer or some other machine. But a great deal of evidence suggests that the two kinds of cognition have been intertwined through evolution. The animals that are adept at using tools, for example, belong to lineages with unusually large brains. It's possible that social cognition drove their expansion.

Robin Dunbar of the University of Oxford and his colleagues have found that the size of mammal brains, relative to body size, tends to be higher in species that are more social (<u>Dunbar 2010</u>). Among primates, the researchers have found a tight positive correlation between the average group size in a species and the fraction of the brain taken up by the neocortex, the region of the cerebral cortex where the most sophisticated information processing takes place (<u>Dunbar 2009</u>; <u>FIGURE 13.24</u>).



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FIGURE 13.24

The social lives of primates may have profoundly affected the evolution of their brains. This graph shows how primate species that live in large groups tend to have a proportionately larger neocortex. (Information from Barrett, Lycett, and Dunbar 2002.)

Most species of primates live their entire lives in a group. They sleep together, search for food together, escape predators together, and sometimes even do battle with other groups of primates together. Living in groups may be better for most primates than living alone, but these groups create a new

arena within which competition arises. Primates struggle to reach the top of the hierarchy in a group, and with that rise often comes reproductive success.

Joan Silk, a primatologist at the University of California, Los Angeles, has analyzed observations of baboon populations in Kenya that span more than two decades. She has found that high-ranking female baboons grow faster, produce healthier infants, give birth at shorter intervals, and generally have a higher lifetime reproductive success than lower-ranking baboons. For baboons, and for many other primates, achieving a high rank is not a matter of having the biggest teeth or the loudest scream. It's more a matter of social connections, of alliances that can last for decades. The most socially integrated baboons, Silk has found, have the highest reproductive success in a baboon society (Silk, Alberts, and Altmann 2003).

These conditions have driven the evolution of primates, making them keen social observers. They can recognize individual members of their own group and remember who is related to whom, and who is allied with whom. They can tell what other members of their group are looking at, and they can use that knowledge to deceive them. A female gorilla will sometimes sneak off with a low-ranking male to mate, and the pair will make only quiet sounds instead of their normally loud mating calls. A kind of social economy has evolved even in monkeys and apes. In 2007, for example, a student of Silk's named Kimberly Duffy reported that a top-ranking male chimpanzee gave lower-ranking males the opportunity to mate with females in his group in exchange for their alliance with him against other males (<u>Duffy, Richard, and Joan 2007</u>).

The larger the group, Dunbar argues, the more powerful the social cognition that is favored. Bigger primate brains are correlated not just with large groups, he finds, but also with other signs of social complexity such as how often primates deceive each other.

Corvids are very social birds, and they also have large brains. But they don't share the correlation between group size and brain size found in primates. In fact, New Caledonian crows, the most cognitively impressive of the corvids, actually live in small family units and rarely interact with other families (Holzhaider, Hunt, and Gray 2010). What they lack in quantity, they make up for in quality. Unlike many bird species, in which the young leave their parents as soon as possible, juvenile New Caledonian crows stay with their parents for over a year. Under these conditions, the juvenile crows

don't have to invent tools on their own. Instead, they can watch their parents make tools and use them. This time for learning is probably essential for the crows because it takes New Caledonian crows more than a year to figure out how to use hooks to get food. In an intriguing parallel, adult chimpanzees also allow juveniles to watch them crack nuts and use other kinds of tools.

The ability that chimpanzees and New Caledonian crows have to learn and come up with new solutions to old problems has also endowed them with something else: culture. The tools used in one population of crows are different in certain ways from the tools made by other populations. Some populations shape pandan leaves into wide probes, while others make them slender. Still others cut a stepped series of notches in the leaves (Hunt and Gray 2003). Chimpanzee technologies also vary from one site to the next. Not all chimpanzees use rocks to smash nuts, for example. Many populations of apes have unique combinations of tools and techniques that neighboring populations lack (Lycett, Collard, and McGrew 2010).

Of course, humans are the supreme toolmakers of the animal kingdom. We are also equipped with the largest brain in the animal kingdom (relative to our body size). And we are tremendously social, existing in societies that can number in the billions. How did human behavior evolve? That question is the subject of the next chapter—where we'll see that the methods scientists use to understand animal behavior can offer profound lessons about human nature, too.

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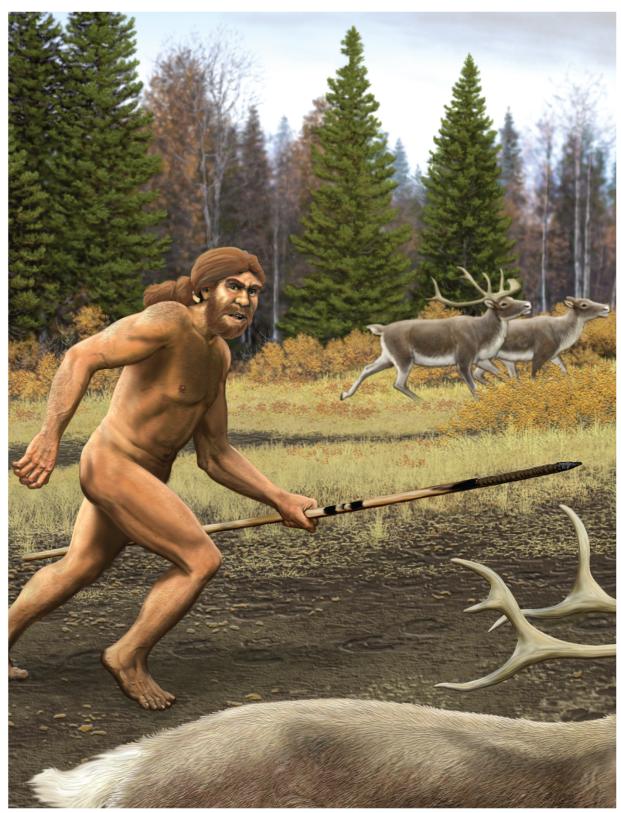
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Carl Buell.

Neanderthals belong to a lineage that diverged from our own lineage 800,000 years ago. Before becoming extinct 28,000 years ago, Neanderthals interbred with *Homo sapiens*, and today billions of people carry a little Neanderthal DNA in their genomes.

The standard uniform for paleontologists is casual. T-shirts, cutoffs, and floppy old hats are common sights around most fossil digs. But in a Spanish cave called El Sidrón, the dress code is decidedly more formal. Fossil hunters regularly suit up in white coveralls, surgical masks, and sterile gloves. And rather than ordinary rock hammers and chisels, the paleontologists at El Sidrón use sterilized blades to dig at the bones, which they quickly put into a freezer. They look less like fossil hunters than characters out of a science fiction movie.



Carles Lalueza-Fox.

FIGURE 14.1

Researchers excavating in a cave in Spain have discovered fragments of DNA in 48,000-year-old Neanderthal fossils. They are comparing the genes to ours to gain clues about how human behavior evolved.

There is, in fact, a science fiction quality to what the researchers are up to: they want to read the genes of an extinct kind of human whose bones lie in the cave. The bones were discovered in 1994 by cave explorers. When the police first came to the cave, they initially assumed the remains belonged to people killed during the Spanish Civil War in the 1930s. But it soon became clear that the bones were much older—in fact, they were 48,000 years old. And instead of belonging to humans like ourselves, they belonged to Neanderthals, a group of hominins that vanished 28,000 years ago (Lalueza-Fox et al. 2012).

Scientists have known about Neanderthals since 1863, when their remains were first discovered in the Neander Valley in Germany. (*Thal* means "valley" in German.) Since then, researchers have found dozens of Neanderthal fossil sites, where they've found not just bones but also tools and other traces of Neanderthal behavior. But in the late 1990s, Svante Pääbo, a geneticist at the Max Planck Institute for Evolutionary Anthropology, pioneered a new way to study Neanderthals: by extracting and analyzing their DNA.

DNA normally breaks down when an organism dies, but under certain conditions, its fragments can survive for tens of thousands of years. Pääbo and his colleagues discovered that some Neanderthal fossils sitting in museums still contained traces of DNA. Even the bones that had been unearthed in 1863 turned out to have genetic material. To find more DNA, the scientists developed new methods for extracting DNA from bones freshly dug up from the ground. To succeed at this audacious task, they have to take every possible precaution to ensure that not even a flake of their skin or a drop of sweat contaminates the fossils with their own DNA.

The precautions start with the masks and suits at places like El Sidrón. But they don't stop there. Researchers ship the frozen remains to Pääbo's ultraclean laboratory in Leipzig. His research team grinds the bones into a powder and gradually removes all the minerals and organic matter until only DNA is left. Then they capture the fragments of DNA and analyze the sequences. In 1997, Pääbo and his colleagues published the sequence of their first sample of Neanderthal DNA. It was only 379 base pairs of mitochondrial DNA—a tiny fraction of the hominin's genes. But in 2010, they unveiled the draft genome of Neanderthals, made up of more than 4 billion nucleotides drawn from material from three different individuals (Greene et al. 2010).

The resurrection of an extinct genome allows scientists to understand these enigmatic people like never before. They are getting a better picture of what Neanderthals looked like, and how their physiology was adaptive for a demanding life hunting big animals. But understanding the Neanderthal genome has another power that's just as important: it allows us to learn things about ourselves.

Neanderthals could hunt elephants and rhinoceroses. They could fashion sophisticated stone tools. They were moved to bury their dead. But the remains of Neanderthals suggest that they couldn't paint a picture. They didn't trade tools over long distances, perhaps because they lacked social skills. In some crucial ways, Neanderthals were different from us. The stages of human evolution that gave us language, symbolic thought, and many other faculties we think make us uniquely human unfolded only after our ancestors split off from the Neanderthals. And now scientists can compare the Neanderthal genome and the genome of living humans to find the mutations that made those adaptations possible.

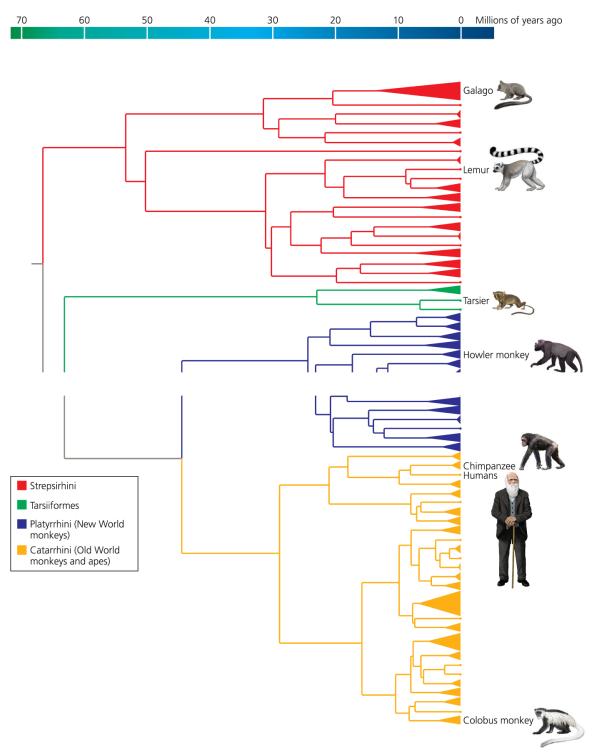
Humans are but one of millions of species alive on Earth today. Each species has its own intriguing evolutionary history, shaped by a vast number of forces. But when we humans look at the history of life, we can't help being obsessed by how our own species came to be. In this chapter, we'll look at the latest consensus about how humans evolved. We've already encountered fragments of this narrative in previous chapters. Here we will synthesize many lines of evidence—from fossils to genomes—to discover our own story.

An Origin among the Apes

When the eighteenth-century naturalist Carl Linnaeus organized all living things into a single classification system, he decided to put humans in the order Primates, along with apes, monkeys, and other species. Linnaeus recognized that humans and other primates shared a number of anatomical traits, from their forward-facing eyes to their gripping thumbs. He made his decision despite knowing it would be a controversial one. "It is not pleasing to me that I must place humans among the primates," he wrote in a letter to a fellow naturalist in 1747, "but I desperately seek from you and from the whole world a general difference between men and simians from the principles of Natural History. I certainly know of none. If only someone might tell me one!" (Linneaus 1747).

In his 1871 book *The Descent of Man*, Darwin argued that the similarities between primates and humans were evidence that they have a common ancestor. He noted that within the primate order, apes are most similar to humans—they are large bodied, have relatively big brains for their bodies, and have a vestige of a tail just like we do. He argued that apes were therefore our closest primate relatives.

Darwin didn't know it then, but our DNA confirms our primate heritage. Mark Springer, an evolutionary biologist at the University of California at Riverside, and his colleagues published the most detailed molecular phylogeny of primates to date in 2012—an evolutionary tree joining 367 out of the 450 estimated species of primates alive today (Springer et al. 2012). By comparing the mutations in each lineage, they could use a molecular clock to estimate the timing of primate evolution. As FIGURE 14.2 shows, they found that the common ancestor of all living primates lived about 68 million years ago.



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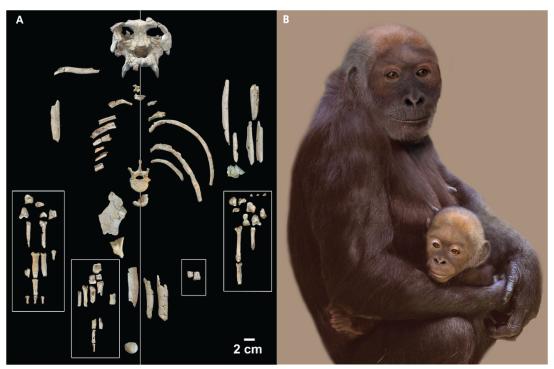
FIGURE 14.2

A recent analysis of DNA from 347 primate species shows how they evolved from a common ancestor some 68 million years ago. Humans are most closely related to great

The primates then branched into their two main lineages. One lineage includes lemurs, found only on Madagascar, and the other includes small tropical primates such as galagos and lorises. The other branch split into new lineages as well. About 40 million years ago, the ancestors of New World monkeys split off from the monkeys of the Old World and the apes. The apes and Old World Monkeys diverged about 30 million years ago. Our own lineage—the hominins—branched off from the ancestors of chimpanzees about 7 million years ago. As with all molecular phylogenies, Springer's primate tree has a range of possible dates for each node. For the human-chimpanzee split, for example, their mean estimate is 6.66 million years ago, with a likely range of between 4.74 and 9.5 million years. As we'll see, that range is a good fit for the fossil record.

Walking into a New Kind of Life

By necessity, Springer and his colleagues could compare only living primates. Ancient DNA survives in appreciable amounts for only tens of thousands of years, not millions. To gain further clues to primate evolution, paleontologists search for extinct species. The earliest primate fossils were tiny, long-tailed creatures that lived in the trees. Starting about 20 million years ago, the fossil record reveals the earliest fossil apes. Found across much of the Old World, these medium- to large-bodied creatures lost their tails; but they still had flexible, strong hands and feet that they could use to grip tree branches (FIGURE 14.3).

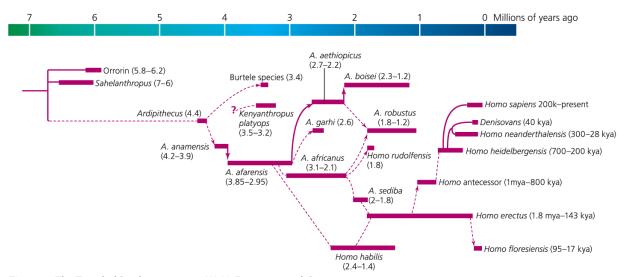


(A) (Left) From "Pierolapithecus catalaunicus, a New Middle Miocene Great Ape from Spain" by Moyå-Solå, Köhler, Alba, Casanovas-Vilar, and Galindo. Science 19 November 2004: Vol. 306, No. 5700 pp. 1339–1344. Reprinted with permission from AAAS. (Right) Meike Köhler. (B) (Right) Pierolapithecus catalaunicus, a New Middle Miocene Great Ape from Spain, Salvador Moyà-Solà1,*, Meike Köhler1, David M. Alba 1,2, Isaac Casanovas-Vilar1 and Jordi Galindo 2, Science 19 November 2004: Vol. 306 no. 5700 pp. 1339–1344 DOI: 10.1126/science.1103094.

Pierolapithecus was a species of ape that lived in Spain 13 million years ago. It was closely related to the common ancestor of humans and other living apes. Like apes today, it lacks the tail found on most monkeys and other primates.

Over time, many of the early ape lineages became extinct. In Europe the apes disappeared completely, while in Asia a few species survived (gibbons and orangutans still cling to existence there today). In Africa, a greater diversity of apes survived. And it's from these African apes that our own lineage—known as hominins—emerged.

In <u>Chapter 4</u>, we examined some of the best-understood hominins to see how they were related to one another (<u>FIGURE 14.4</u>). The full fossil record of hominins is much richer, but also more challenging to interpret. One species is known only from part of its foot, another only from fragments of its skull. These fossils usually contain enough information for paleoanthropologists to be certain that they belonged to hominins and not to some other ape. But they have different views about which hominins are most closely related to which others. <u>Figure 14.4</u> shows the current state of our understanding of the fossil record—an understanding that improves with each new discovery.



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FIGURE 14.4

The hominin fossil record continues to grow. It remains challenging to determine which species are most closely related to each other. This chart shows the current consensus—

solid lines show wellsupported links, while dotted ones are more contentious. (Information from <u>Harmon, 2013</u>.)

Despite these uncertainties, some important patterns do emerge from the hominin fossil record—patterns that can help us understand our origins. The hominin lineage emerged as Earth's climate was undergoing some dramatic changes, for example. The average temperature of the planet dropped, and Africa received less rainfall. Lush tropical forests no longer covered the continent like a thick blanket. Drier woodlands and even some grasslands began to expand. Instead of steady weather patterns throughout the year, these new habitats experienced seasons of rain and drought, making supplies of food less predictable (Klein 2009).

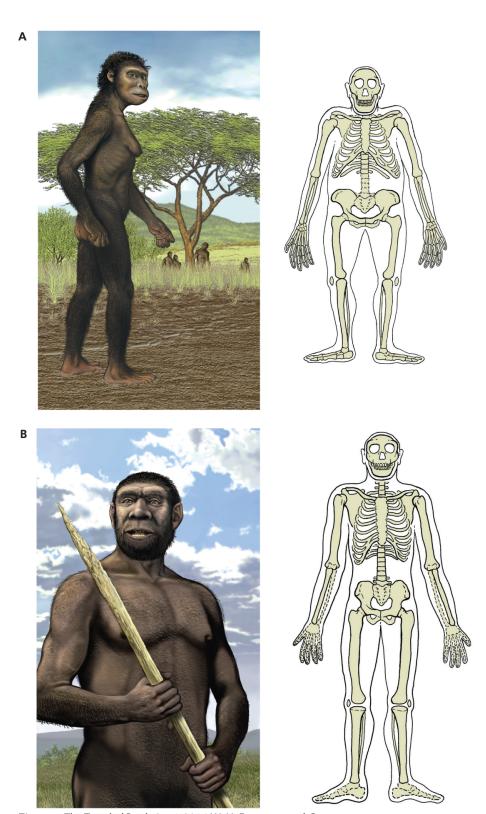
The changing climate was likely responsible for the extinction of most early ape lineages. Of the five species of great apes alive today, four of them are found in tropical rain forests. Orangutans survive in Indonesia, while gorillas and the two species of chimpanzees—the common chimp and the bonobo—survive in Africa's forests. Their lineages remained adapted to forests, and today that dependence is helping to push them toward extinction as humans speed up the destruction of their forest homes and hunt them for food (Stanford 2012).

Hominins, on the other hand, adapted to the new ecosystems. The places in East Africa where the best hominin fossil sites are located fluctuated between grasslands and sparse woodlands between 7 and 2 million years ago before switching over to savannas (Cerling et al. 2011). Becoming bipedal may have been a key transition in the adaptation of hominins to their new environment (Lieberman 2011). As we discussed in Chapter 4, some of the oldest hominin fossils have traits that suggest they had at least some capacity to walk upright.

Scientists are exploring a number of hypotheses for why natural selection favored bipedalism in hominins. Kevin Hunt of Indiana University and his colleagues have argued that it allowed hominins to do a better job of gathering food. The more stable their bodies, the more fruit they could collect from trees. Such an origin might explain why early hominins still had broad pelvises. Such a skeleton might provide solid footing for stretching to reach

fruit, although it meant that early hominins would be inefficient walkers (<u>Hunt 1994</u>).

As hominins became more adapted to open grasslands, other selective pressures may have driven them toward more efficient walking (FIGURE 14.5). Without the protection of a forest canopy, hominins would face a new challenge of intense heat on the savanna. Peter Wheeler, of Liverpool John Moores University, argues that an efficient upright stride would have helped hominins to stay cool (Wheeler 1991). By standing upright, they exposed less of their skin to the sun, and they could hold their heads up in cooler, breezier layers of air.



Zimmer, The Tangled Bank, 2e, © 2014 W. H. Freeman and Company a,b: Carl Buell.

A: Australopithecus afarensis lived between 3.85 and 2.95 million years ago. It was a small-brained, long-armed hominin that may have spent part of its time walking and part of its time in the trees. B: By 1.8 million years ago, hominins such as *Homo erectus* had evolved. They were taller and had long legs and other adaptations that made them efficient walkers and runners.

Teeth and Tools

Between 4 and 2 million years ago, hominins remained fairly small, and still had chimp-sized brains. Yet these hominins—collectively known as australopiths—displayed a remarkable diversity. Some were slender, with teeth that were much reduced from those of their ape ancestors. Others had a more gorillalike build, with wide jaws and broad teeth. These differences were likely the result of the food they specialized in eating—a diverse selection that included seeds, roots, insects, fruits, and perhaps even grasses.

But teeth could get a hominin only so far. The African grasslands and woodlands swarmed with great herds of grazing mammals, for example. Lions could take down that game with their dagger-like teeth and claws and feast on the protein-rich muscle of their prey. Strong-jawed hyenas could crack open the bones to feed on the marrow inside. The small-bodied, small-toothed hominins couldn't take advantage of this abundant source of food. But that began to change when they started making stone tools.

In the last chapter, we saw how a few animals, such as chimpanzees, are adept toolmakers. Hominins likely started out with that same ancestral capacity, possibly making sticks to dig out termites or using rocks to crush nuts. But hominins evolved the mental capacity to make more powerful and versatile tools that other animals couldn't craft.

In Ethiopia, Shannon McPherron of the Max Planck Institute for Evolutionary Anthropology and his colleagues have found what may be the oldest evidence of these tools: 3.4-million-year-old bones of large mammals with distinctive cut marks (McPherron et al. 2010; FIGURE 14.6). Some skeptics won't be persuaded until McPherron and his colleagues find the stone tools that made those marks. If McPherron turns out to be right, it's most likely that some species of hominin alive 3.4 million years ago was fashioning tools for use in butchering animals for its meat. McPherron and his colleagues have also found cracked bones at the site; hominins may have broken them to eat the protein-rich marrow inside.



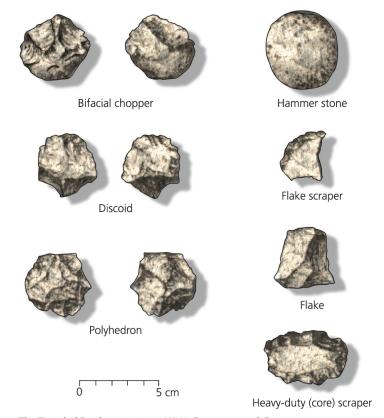
Shannon McPherron.

FIGURE 14.6

Mammal bones discovered in Ethiopia bear distinctive cut marks. In 2010, researchers published these bones and declared them the earliest evidence of stone tool use by hominins.

The oldest stone tools that are generally accepted, found at another site in Ethiopia called Gona, date back 2.6 million years. The tools were chipped to make them good for cutting, and near the tools scientists have found bones with cut marks on them. The hominins that made the tools at Gona displayed great care and planning. They picked out certain rocks from riverbeds and carried them for miles before stopping to manufacture them into tools (Stout et al. 2005).

Over the next million years or so, hominins in other parts of Africa continued to make similar tools. The style is known as the Oldowan, named for a region of Kenya where some of the first tools of this period were discovered. Hominins used round "hammer stones" to chip off bits of other rocks and then used the smaller rocks to make a range of tools, from choppers to scrapers (FIGURE 14.7). They may have used the tools to carve off meat and hammer open bones.



Zimmer, The Tangled Bank, 2e, © 2014 W. H. Freeman and Company

Hominins developed a style of toolmaking called Oldowan. The shapes of the tools show how they were made, by striking off flakes from large, round rocks. Hominins used tools to scavenge meat and possibly to fashion wooden tools. (Information from Boyd and Silk 2009.)

There is some evidence that hominins used their tools to carve sticks and bones, perhaps to dig up tubers and break open hard termite nests. Toolmaking, in other words, is a form of ecological specialization that allowed hominins to find calories and protein that many other animals couldn't get. Over the past 2.6 million years, we've become ever more dependent on our technology for survival, and our technology continues to grow more powerful.

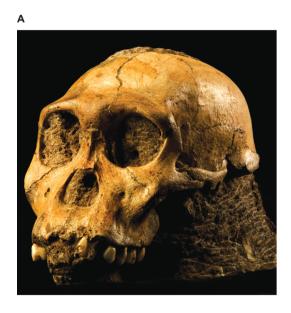
The Arrival of *Homo*

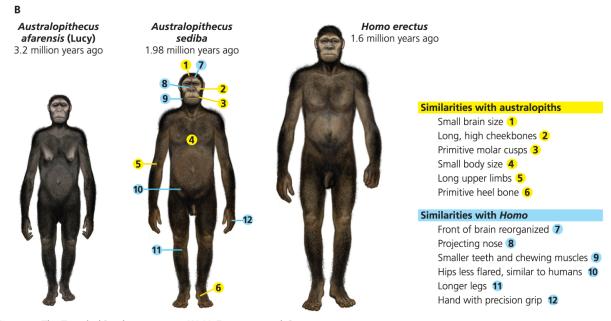
When Linnaeus first classified our own species, he placed it in its own genus, *Homo*. But starting in the mid-1900s, scientists began to add other species to it as well. These other *Homo* lineages share a number of traits that unite them with our own species but are not found in the australopiths. *Homo erectus*, for example, was a species that lived from 1.89 million to 143,000 years ago. It was tall and had a slender pelvis, long legs, and a brain much larger than those of earlier hominins.

The transition from australopiths to *Homo* is a remarkable one. An important clue to how it happened came in 2008, when Lee Berger, a paleoanthropologist at the University of Witwatersrand, was taking a walk with his nine-year-old son Matthew. Father and son were strolling near a cave called Malapa in South Africa when Matthew ran after their dog into a stand of tall grass and tripped over a log. When he stood up, he was holding a fossil clavicle.

Berger and his colleagues explored the cave further and discovered the remains of several individuals from a hominin species that lived between 1.95 and 1.78 million years ago. Together, the fossils represented a new species, which Berger and his colleagues dubbed *Australopithecus sediba* (Berger et al. 2010).

A. sediba proved to have a remarkable mix of traits (FIGURE 14.8). Some traits were ancestral, found in other australopiths. Others had previously been found only in Homo. It had long, australopith-like arms, for example, but its hands were short, much like our own. Thanks to its long legs, it stood about 1.5 meters tall. But its ankle bones still looked like those of an australopith. It had a projecting nose, similar to ours, but still had a tiny brain. Berger and his colleagues argue that A. sediba was either the ancestor of Homo or one of the closest arelatives to the clade. It shows that even before Homo had evolved, some of the hallmarks of Homo already existed.





Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Photo by Brett Eloff, courtesy of Lee Berger and the University of the Witwatersrand. B: These reconstructions illustrate the combination of traits in A. sediba, some of which it shares with australopiths and some of which it shares with *Homo*.

A: In 2010, Lee Berger of the University of Witwatersrand and his colleagues described this skull and other fossil material from a new species of hominin. They named it *Australopithecus sediba* and estimated that it lived at some point between 1.95 and 1.78 million years ago.

The transition from australopiths to *Homo* marked an important change in the natural history of hominins. *Homo erectus* and other early *Homo* fossils have lost the adaptations for tree climbing found in earlier hominins. Their hands, for example, no longer had special muscles for hanging from tree branches. Their long legs and straight feet now allowed them to walk efficiently, no longer using extra energy in moving their limbs out to the sides. Daniel Lieberman of Harvard and Dennis Bramble of the University of Utah have pointed to some subtle anatomical details that suggest early *Homo* could also run for long distances—something that they argue australopiths could not do. It's possible that *Homo* began to run in order to find food—either by hunting or by reaching animal carcasses before hyenas or other scavengers got there (Lieberman and Bramble 2007).

When these hominins reached their prey, they brought with them new kinds of tools. They created large "hand axes," for example, by carefully chipping away large rocks to create teardrop shapes (FIGURE 14.9). These new tools are known as Acheulean technology, named after the site in France where they were first discovered. Scientists debate exactly what early *Homo* used hand axes for. But whatever their use, they speak of a major transition in how hominin brains worked. The transition meant that *Homo* had much more delicate control of its hands and was able to make more detailed plans for fashioning an ordinary rock into elaborate creations.



John Reader/Science Source.

FIGURE 14.9

Between 1.5 million and 200,000 years ago, hominins in Africa, Asia, and Europe made large, teardropshaped stone tools. They probably used these so-called hand axes to

butcher animals, cut wood, and gather roots and other plant foods.

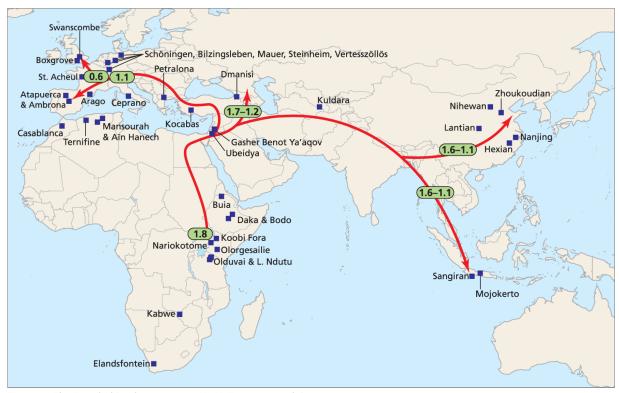
Yet early *Homo* still had a mind very different from our own. They were capable of developing new technology, but at a pace that to us seems unimaginably slow. *Homo* continued to use Oldowan tools as well for half a million years, until about 1.2 million years ago. And the "new" Acheulean tools did not rapidly give way to new designs, the way our cell phones become obsolete in a matter of months. They hardly changed shape during the period from their first appearance 1.7 million years ago to their final disappearance only 100,000 years ago.

The narrow rib cage of *Homo* also suggests that these changes in locomotion and technology led to a new kind of digestive system. Mammals that eat a lot of plant matter have long stretches of intestines where the tough molecules in the food can slowly break down. The flaring shape of australopith rib cages suggests a massive digestive system for eating mostly plants. The narrow rib cage of *Homo erectus*, on the other hand, suggests it had a shorter digestive system—perhaps one that was adapted to a diet with more meat or more energyrich tubers.

The anthropologist Leslie Aiello proposed that this shift was crucial to the expansion of the hominin brain. Brains require huge amounts of energy; one out of every five calories we eat goes to fueling our brains. Aiello argues that as the guts of hominins shrank, they were able to direct energy away from maintaining their intestinal tissues and toward an expanding brain (Aiello and Wheeler 1995).

Out of Africa, the First Time

The emergence of *Homo* also marked the first time that hominins left the continent where they had evolved (FIGURE 14.10). *Homo erectus* spread as far east as Indonesia. Other species arrived in Europe by 1 million years ago. Most of the early *Homo* fossils found outside of Africa are tall and slender, but some fascinating exceptions to the rule have turned up over the past decade. In the Republic of Georgia, for example, researchers discovered a number of fossils of hominins that date back 1.8 million years (Lordkipanidze et al. 2007). *Homo georgicus*, as they're sometimes called, have many hallmarks of *Homo*, but they were also very short and had a small braincase, measuring just 600 cubic centimeters in volume—less than half the size of our own.

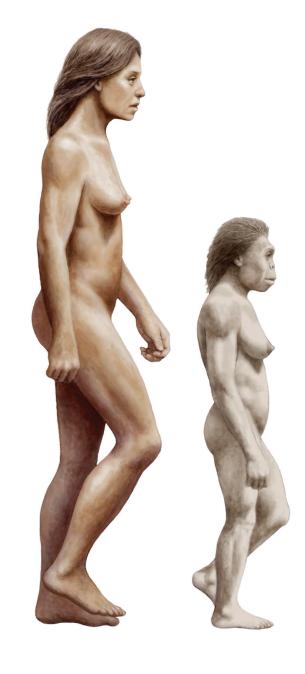


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Hominins first emerged out of Africa 1.8 million years ago. The lineage that would give rise to *Homo sapiens* remained in Africa. (Information from <u>Klein 2009</u>.)

In 2004, paleoanthropologists found even more striking fossils on the Indonesian island of Flores. The fossils, ranging in age from 90,000 to 18,000 years old, belonged to a hominin that stood just 1 meter tall; they had skulls of just 417 cubic centimeters (FIGURE 14.11). The scientists who found these fossils dubbed them *Homo floresiensis* (Aiello 2010).





Left: Carl Jungers; right: Mauricio Anton/Science Source.

Left: This fossil skeleton belongs to *Homo floresiensis*, which lived on the Indonesian island of Flores until 18,000 years ago. *Right:* In this reconstruction, a human woman (left) is shown next to a reconstruction of *H. floresiensis*, which stood only 1 meter tall and had a chimpanzee-sized brain.

Scientists are sharply divided about the origin of these small Asian hominins. Some scientists have argued that *Homo floresiensis* was actually just a population of *Homo sapiens*. Modern humans have evolved to have short stature in several regions of the world. But living pygmies still share many traits found only in living humans—including a large brain. *Homo floresiensis*, by contrast, had a small brain and also lacked a number of other defining traits. In the face of this evidence, some researchers have proposed that the unusually small Flores fossil skull was the result of disease—perhaps a genetic disorder or a deficiency in iodine.

Others have argued that *Homo floresiensis* is not a branch of *Homo sapiens*, but of *Homo erectus*. At some point hundreds of thousands of years ago, a population of *Homo erectus* in Indonesia came to the island of Flores and then evolved its small stature. A similar explanation has been offered for the origin of *Homo georgicus*.

But others have offered a radical alternative. Perhaps some australopiths left Africa first. If that's true, then efficient bipedal walking could not have been a requirement for leaving Africa. These exciting possibilities have spurred scientists to look even harder for early hominin fossils outside of Africa.

Parallel Humans

About 600,000 years ago, the fossil record marks another important transition in hominin evolution. Hominin fossils from Africa that date back to around this time show clear signs of diverging from *Homo erectus*—including the size of their brains. These hominins (*Homo heidelbergensis*) had brains measuring about 1200 cubic centimeters, just 200 cubic centimeters shy of the typical size of brains in living humans.

These hominins once again expanded out of Africa to Asia and Europe. As they spread, they left behind some of the earliest evidence that hominins could hunt. On the island of Jersey, in the English Channel, the fossils of rhinos and other big mammals have been found at the bottom of cliffs, where they show signs of having been butchered. It's likely that they were killed by hominins who chased them off a precipice and then finished them off at close range. More evidence for hunting comes from the tools they made. German archaeologists digging in the remains of an ancient lake discovered wooden spears dating back 400,000 years. Sharpened like javelins at both ends, the spears were between 2 and 3 meters long (Thieme 1997). Not far away from the spear site, the archaeologists found the butchered bones of wild horses. It's possible that *Homo heidelbergensis* drove these horses into a lake and then killed them with their spears.

The climate of Europe could be harsh at this time. Ice ages brought glaciers over the northern edge of the continent and turned the southern regions into dry wastelands. Over time, natural selection altered the bodies of these European hominins. The legs became stubby, the chests wider, the bodies more muscled. By about 300,000 years ago, fossils indicate the European hominins had become markedly different from the more slender hominins in Africa. This European lineage of hominins eventually evolved into Neanderthals (FIGURE 14.12).



Blaine Maley.

Neanderthals (left) had stouter, more muscular bodies than modern humans (right). Now that the Neanderthal genome has been sequenced, scientists are cataloging their genetic differences as well.

Neanderthals had brains at least as big as our own. The isotopes in their bones suggest a diet rich in meat, and their fractured bones indicate that they had to withstand a lot of abuse to hunt their food. Despite the lack of evidence that Neanderthals could paint images or make sculptures, they still left behind many traces of sophisticated behavior. Their tools were advanced, and they colored shells with pigment and drilled holes into them,

perhaps to string them on necklaces (FIGURE 14.13). About 28,000 years ago, though, the last traces of Neanderthals vanish from the fossil record.

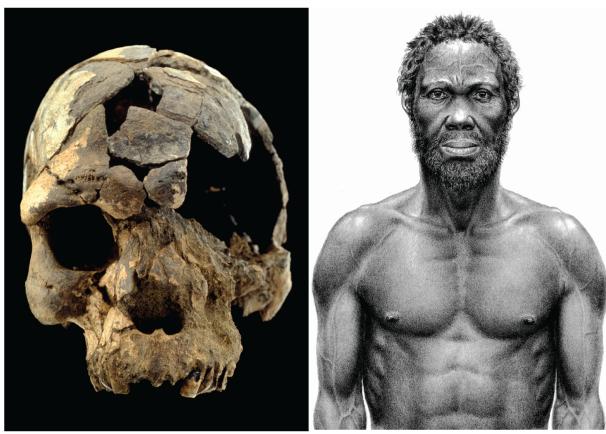


Jaao Zihao.

FIGURE 14.13

Shells found at a Neanderthal site were drilled and painted. This photograph shows two sides of a single piece of shell, painted white on one side (right). Scientists suspect Neanderthals were these shells as ornaments—an activity that may reveal clues to their minds. (Information from Zilhäo et al. 2010.)

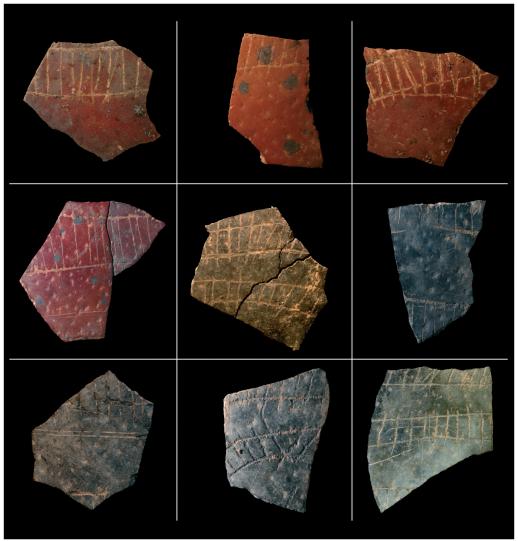
Neanderthals weren't the only species to diverge from *Homo heidelbergensis*, however. Neanderthals evolved in Europe and Asia; in Africa, *Homo heidelbergensis* gave rise to a new species—our own. As we saw in <u>Chapter 4</u>, the oldest fossil that shows clear signs of belonging to *Homo sapiens*, a skullcap found in Ethiopia, dates back to 200,000 years ago. A better-preserved skull from Ethiopia, dating back to 160,000 years ago, has been dubbed *Homo sapiens idaltu* (White et al. 2003; FIGURE 14.14).



Left: David L. Brill; right: © 2003 Jay H. Matternes.

Homo sapiens idaltu, a subspecies of modern humans, lived in Ethiopia 160,000 years ago.

The artifacts that *Homo sapiens* left behind in Africa document an accelerating pace of change. Across the continent, for example, tools acquired a local flavor. But humans also began trading their tools, and some of them ended up hundreds of kilometers from where they had been made. Humans in Africa began to show signs of self-expression, such as pierced snail shells that might have gone on a necklace. They also began making abstract decorations, such as geometrical patterns on ostrich eggs used for water containers (FIGURE 14.15).



Pierre-Jean Texier.

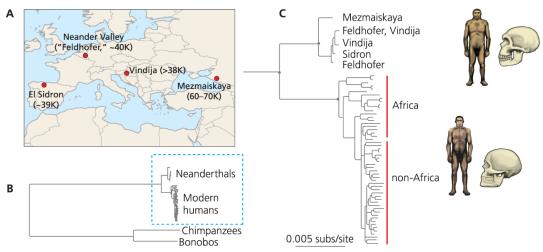
After *Homo sapiens* arose in Africa, our ancestors displayed an increasing level of creativity and self-expression. In South Africa 60,000 years ago, humans made water containers out of ostrich eggs and decorated them with geometrical patterns.

The descendants of these early humans would later spread across Africa and then the world. By the time they were done, several other hominin species would become extinct. We humans, *Homo sapiens sapiens*, would become the last surviving species in our 6-million-year lineage (Stringer 2012).

Human Evolution: The Genes Speak

Over the past 30 years, scientists have probed human DNA for clues to human evolution. In <u>Chapter 7</u>, for example, we saw how Sarah Tishkoff has produced a molecular genealogy of living humans. By analyzing the variation in the DNA of living humans, Tishkoff found that most genetic diversity in living humans is found in Africans. Her research supports the view that *Homo sapiens* evolved in Africa and then expanded to other continents. Svante Pääbo and his colleagues are expanding this approach by salvaging a growing supply of ancient DNA from extinct hominins.

The picture that has emerged from these studies has grown deeper and richer in ways no one could have predicted. At first, Pääbo and his colleagues could compare only short sequences of DNA from Neanderthals to those of living humans. FIGURE 14.16 shows a phylogeny that emerged from this research. It shows all living humans sharing a recent common ancestor, but the lineage does not include Neanderthals. Thus the lines of evidence from both modern and ancient DNA agree that Neanderthals and humans represent two separate lineages of *Homo* descending from a common ancestor. As the fossil record clearly shows, Neanderthals became extinct 30,000 years ago. *Homo sapiens*, meanwhile, spread out of Africa and would ultimately populate the entire planet.



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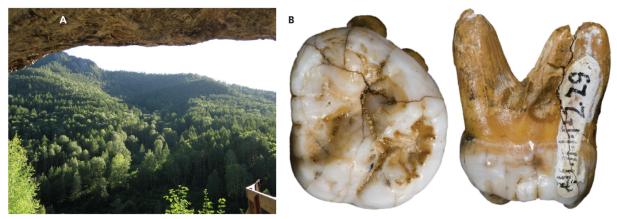
Using new techniques to capture fossil DNA, scientists were able to compare genomes of Neanderthals to those of living humans. A: This map shows sites where Neanderthal DNA has been recovered, ranging in age from 38,000 to 70,000 years old. B: Svante Pääbo and his colleagues found that Neanderthals and modern humans formed a single clade. C: If we zoom in on the box in B, we can see that the ancestors of all Neanderthals (top) diverged from the ancestors of all living humans. Humans and Neanderthals thus represent two distinct hominin populations—or arguably even two hominin species (Information from Briggs et al. 2009.)

In 2010, Pääbo and his colleagues published the first draft of the Neanderthal genome (Green et al. 2010). The scientists made another comparison of Neanderthal and human DNA, but this time the comparison was far more detailed. They identified alleles for each gene in Neanderthals and various human populations. They compared those alleles to estimate how much time had passed since they had diverged from an ancestral gene. Most alleles had a history consistent with Neanderthals and humans belonging to separate lineages. But a small portion of Neanderthal alleles presented a confusing pattern.

These confusing Neanderthal alleles were more closely related to European and Asian alleles than they were to African ones. If Neanderthals were a completely separate lineage, their alleles should have been equally related to all human populations.

To explain this pattern, Pääbo and his colleagues propose that humans and Neanderthals had interbred. After some humans left Africa, they made contact with Neanderthals, and some hybrid children were the result. Through these hybrid children, Neanderthal alleles were able to spread into the human population. Today these alleles make up a small portion of the human genome pool. Pääbo and his colleagues estimate that the people of Europe and Asia have genomes that are on average 2.5 percent Neanderthal.

Ancient DNA has also revealed what may have been an entirely separate species of hominin that existed alongside our own (Reich et al. 2010). In a cave known as Denisova in the Altai Mountains of Siberia, Russian scientists found a 50,000-year-old finger bone from a young girl; the bone turned out to be packed with DNA (FIGURE 14.17). Pääbo and his colleagues sequenced the entire genome contained in the bone cells and found that it belonged to an entirely new lineage of hominins, which they call the Denisovans.



Left: Ann Gibbons; right: Max Planck-Institute of Evolutionary Anthropology.

FIGURE 14.17

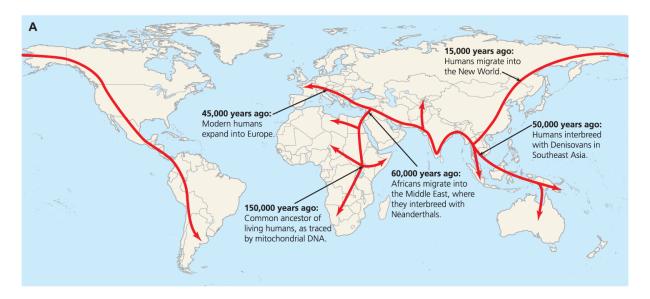
A Siberian cave called Denisova (A) has yielded 40,000-year-old hominin fossils including a tooth (B). A bone fragment in the cave preserved enough DNA to reconstruct an entire genome, which turned out to be different from both humans and Neanderthals. Scientists have dubbed this hominin lineage the Denisovans.

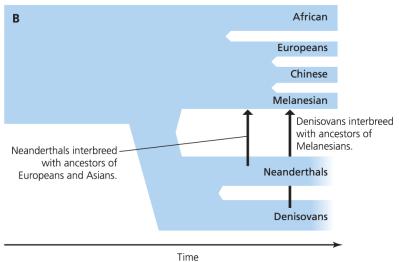
A comparison of the genomes of Denisovans, Neanderthals, and living humans indicate they all share a common ancestor that lived some 800,000 years ago (Meyer et al. 2012). The ancestors of humans then branched off

from the ancestors of Neanderthals and Denisovans. About 400,000 years ago, the Neanderthals and Denisovans then diverged. It's possible that the ancestors of these two lineages emerged from Africa and then split: the hominins traveling westward evolved into Neanderthals, and the ones going eastward evolved into Denisovans.

We still know next to nothing about the fossil record of Denisovans. Along with the fingertip that yielded their genome, the Russian scientists found an isolated tooth that contained mitochondrial DNA showing it to be Denisovan as well. (The nuclear DNA in the tooth was too degraded to rescue.) It's possible that paleoanthropologists have already unearthed Denisovan fossils but incorrectly classified them as human or Neanderthal. In the future, it may be possible to correct these errors and gradually reconstruct Denisovan morphology.

But it is already clear that these ghostly hominins have left their mark on millions of people today. Denisovan alleles are present in populations in Australia, New Guinea, and the Philippines (Reich et al. 2011). It's intriguing that the people who carry Denisovan alleles live thousands of miles to the southeast of the Denisova cave. Pääbo and his colleagues propose that the range of the Denisovans spread from Siberia to Southeast Asia. Humans expanding out of Africa may have encountered the Denisovans in Southeast Asia some 50,000 years ago. They interbred at some point, acquiring Denisovan alleles, which they then carried as they sailed to Australia and neighboring islands (FIGURE 14.18).





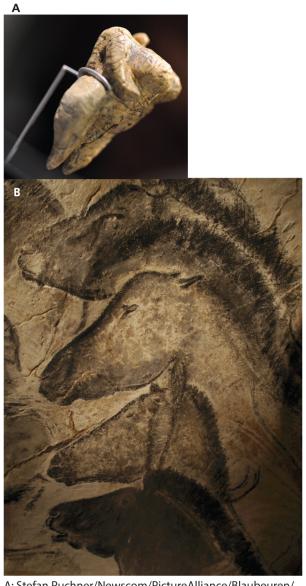
Zimmer, The Tangled Bank, 2e, © 2014 W. H. Freeman and Company

A: Studies on ancient hominin genomes indicate that Neanderthals interbred with humans that emerged from Africa, and Denisovans interbred with humans in south Asia. These humans then spread to Australia and the Pacific. Denisovan DNA is no longer present in south Asia, because the original human populations were replaced by people who did not descend from human–Denisovan hybrids (Adapted from Stringer 2012.) B: By combining information from DNA with the fossil record, scientists can reconstruct the spread of *Homo sapiens*. Humans expanded across Africa between 200,000 and 100,000 years ago and then emerged out of Africa about 60,000 years ago. As they expanded, they interbred with other populations of hominins. (Information from Reich et al. 2010.)

Scientists are now exploring the fate of the Neanderthal and Denisovan DNA after humans acquired it. Much of it has disappeared, due to either genetic drift or negative selection. But scientists are also pinpointing genes that have experienced natural selection. Michael Hammer of the University of Arizona and his colleagues have been comparing alleles of a gene called *STAT2*, which encodes a protein that's part of the signals that activate our immune system (Mendez et al. 2012). They found that a Neanderthal allele for *STAT2* is present in Europeans and Asians, but not in people from sub-Saharan Africa. Intriguingly, it is present in most populations at only 5 percent, but in New Guinea 54 percent of people studied carried it. It's possible that the Neanderthal allele for *STAT2* provides better protection to the diseases people face in New Guinea.

Evolving a Human Brain

The human brain is arguably the most complex structure on the planet, if not the universe. It allows us to thrive across the entire planet, to use computers and other sophisticated technology, to use full-blown language to share subtle stories and concepts (FIGURE 14.19). Any account of human evolution must address how this remarkable organ came to be.



A: Stefan Puchner/Newscom/PictureAlliance/Blaubeuren/Baden-Württemberg/Germany; b: Javier Trueba/ MSF/Science Source.

Figurative art begins to appear in the fossil record roughly 40,000 years ago. A: A female sculpture dating back 35,000 years ago, found in Germany. B: Animal paintings on the walls of caves in Lascaux, France, dating back 20,000 years ago. The emergence of symbolic art marks the emergence of human brains that functioned like ours do today.

But deciphering the evolution of the human brain is difficult. Brains, after all, never fossilize. Yet scientists have developed hypotheses by gathering

other kinds of information from fossils, genes, and other sources. Much of the human brain, for example, is devoted to processing vision. A mouse or a dog, by contrast, has a brain far more specialized for smell. To understand this difference, we have to reach back 40 million years. And we can explore that period of our evolution by analyzing our genes.

Our genes record a shift from smell to sight. All tetrapods use the same family of genes to produce odor receptors on the ends of neurons that grow inside their noses. Like other genes, these olfactory receptor genes were sometimes accidentally duplicated. Afterward, some of the duplicated genes evolved to become sensitive to different kinds of odors, while others were disabled by mutation. Disabled olfactory receptor genes lingered on as pseudogenes in many lineages, and many of them were deleted from the genome in others. Mammals that depend on their sense of smell have large families of olfactory receptor genes. Mice, for example, have 1391 olfactory receptor genes, and 508 (about 36 percent) of them are pseudogenes. Primates, by contrast, have far fewer olfactory genes and a higher proportion of pseudogenes. Humans have 802 olfactory genes, but 415 of them—more than half—are pseudogenes (Niimura and Nei 2007).

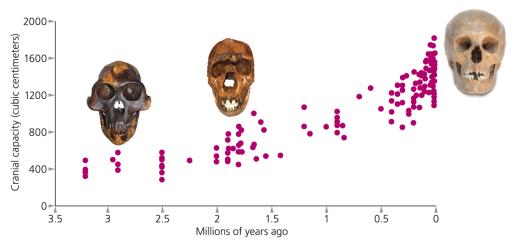
A number of studies indicate that our primate ancestors shifted from smell to sight because of a shift in their diet. Old World monkeys and apes eat mainly leaves and fruit, and they depend on their ability to judge when a particular fruit or leaf is ready to be picked. It turns out that Old World monkeys and apes also share a duplicated opsin gene that other primates lack, and this gene duplication gives them better vision in the red and orange region of the light spectrum. This trichromatic color vision helps detect developing leaves with high nutritional content from a greater distance (Lucas et al. 2003).

As the climate changed and fruits became scarce, the ancestor of apes and Old World monkeys apparently came to rely more on vision to find food and less on smell.

This shift toward vision also altered the social lives of primates. Many other mammals communicate to each other with a language of odors. The molecules that waft from a newborn lamb enter its mother's nose and trigger changes in her brain. She will recognize her lamb by smell until she has finished nursing it. Primates respond more emotionally to the sight of their fellow primates than to their smell, however. This transition brought with it

the evolution of a new kind of face. New arrangements of facial muscles evolved that allowed primates to make a much wider range of expressions. New regions of the brain also specialized in recognizing the faces of other primates and understanding what kind of face they were making (<u>Burrows</u>, <u>Waller</u>, and <u>Parr 2009</u>).

As we saw in the last chapter, Robin Dunbar argued that social evolution drove the expansion of the primate brain. As larger group sizes evolved in some primate species, a larger neocortex evolved. Although early hominin brains have not survived, scientists have been able to estimate their size by measuring the size of fossil braincases (FIGURE 14.20). For the first few million years of hominin evolution, hominin brains were about the size of chimpanzees' brains. But about 2 million years ago, hominin brains began to expand and continued to do so until about 100,000 years ago. If Dunbar's hypothesis is correct, hominin group sizes were increasing, until they reached about 150 individuals.



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Left: Sabena Jane Blackbird/Alamy Middle: Homo ergaster skull reconstruction, Bone Clones®models www.bonesclones.com, Right: DoctorKan/Shutterstock.

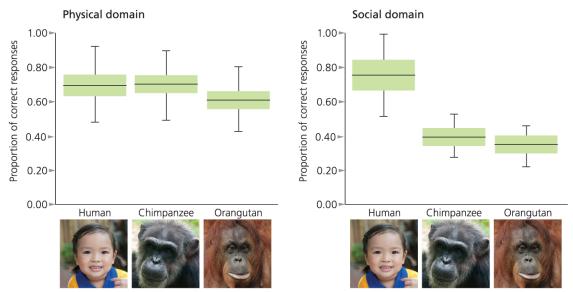
FIGURE 14.20

Early hominins had brains about the size of a chimpanzee's. About 2 million years ago hominin brains began to increase, and after 1 million years their growth accelerated. Today humans have brains about three times the size of those of the earliest hominins. (Information from Nicholas Matzke, http://www.ncse.org.)

This hypothesis suggests that the fundamental difference between humans and other apes is that we became "ultra-social." In 2007, Esther Herrmann and her colleagues at the Max Planck Institute for Evolutionary Anthropology tested this ultra-social hypothesis by comparing the mental skills of 105 two-year-old children against those of 106 chimpanzees and 32 orangutans (Herrmann et al. 2007).

Herrmann gave the children and the apes an identical series of tests. Some of the tests measured their understanding of space, quantities, or physical causes and effects. To test their spatial memory, for example, she put a toy or a piece of food under one of three cups and then let her subjects try to pick the right cup. Herrmann also tested the children and apes for social cognition. For instance, she showed her subjects how to get a toy or a piece of food out of a plastic tube, and then she gave them another tube. If they could learn by observing, they would be able to open it by themselves.

The ultra-social hypothesis predicts that children rapidly develop social skills such as learning from others and understanding what other people know or don't know. They shouldn't be particularly adapted for understanding math—at least no more than other apes. Of course, children would later go on to understand math far better than apes, but only after they had developed the social skills that they will then use to learn. When Herrmann and her colleagues had finished running the test and tallying the results, they discovered that, as predicted, children did no better than apes on the physical tests. On the social tests, however, they did significantly better. This study offers strong support to the hypothesis that what makes us unique is our sociality (FIGURE 14.21).



Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Left: human: tratong/Shutterstock; Middle; chimpanzee: EPG_EuroPhotoGraphics/Shutterstock; Right: orangutan: Muhammad Izzat/Shutterstock.

Toddlers understand physics about as well as juvenile chimpanzees and orangutans. But they fare much better on tests of social understanding. This difference is predicted by the hypothesis that social intelligence drove the evolution of the human brain. (Information from Herrmann et al. 2007.)

Our ultra-social brains evolved through mutations to the genes that encode the molecules that make them up. By comparing the human genome to the genomes of Neanderthals, Denisovans, chimpanzees, and other primates, scientists are starting to develop a catalog of those genes. But some of the most important changes may not be to the protein-coding region of the genes. As we saw in <u>Chapter 8</u>, shifts in the regulation of genes can also cause dramatic evolutionary changes. Recent studies of human brain evolution point to regulation as being particularly important (<u>Haygood et al. 2010</u>).

Ralph Haygood of Duke University and his colleagues have investigated one example of the evolution of gene regulation in the human brain. Cells use glucose as fuel, and to draw it in from the bloodstream, they build transporters on their surface. How many transporters they build is determined by the regulation of the glucose transporter gene.

Haygood and his colleagues have found that glucose transporters are present in greater numbers in the human brain than in chimpanzee brains—but in fewer numbers in muscles in humans than in chimpanzees (<u>Fedrigo et al.</u> 2011). These changes in gene expression may have helped to fuel a bigger brain by shunting more glucose to the brain and away from other parts of the body.

The Language Instinct

As humans evolved into ultra-social apes, an ultra-social way to communicate also evolved: language. As far as we know, no other species can communicate with full-blown language—a system made up of sounds, gestures, or written symbols that convey information not just about what's immediately in front of us, but what lies in the distant past, in the far-off future, or in a world that never will be.

Language, many scientists argue, is at the core of human nature. To use it, we have to be able to understand abstract concepts, instead of just using labels for obvious things like snakes or birds. Language lets us do things other animals cannot do, such as make complex plans together and gain a deep understanding of the inner lives of other humans (Pinker 1995).

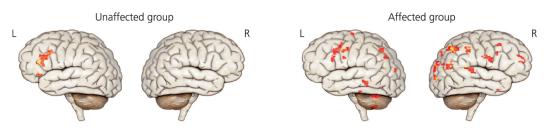
There are more than six thousand languages on Earth, each the product of a particular culture with a particular history. But underlying this staggering diversity, languages have a lot in common. All spoken languages are based on sets of sounds, which can be combined into thousands of words, which in turn can be combined into a countless number of sentences. Words cannot be tossed randomly into a sentence in any language; they all share some basic rules of grammar and depend on syntax for their meaning.

Despite language's complexity, children don't need to attend a linguistics class to learn how to speak. They quickly pick up the rules of grammar for themselves in the first few years of life. Along with having a capacity for learning the rules of language, our brains are also well adapted to hearing speech. When we listen to someone speaking, the network of brain regions that becomes active is not the same as the one we use to listen to ordinary sounds. Certain kinds of brain damage cause "word deafness," leaving people unable to understand speech but still able to hear other sounds.

In considering all of this evidence, scientists suggest that language in humans is an adaptation that was shaped by natural selection. But it was not until the discovery of *FOXP2* (page 177) in 1990 that scientists began to uncover some of the genes underlying our capacity for language. British

researchers pinpointed the *FOXP2* gene while studying a family in which many members had trouble speaking and understanding grammar.

Some of the family members took part in a study run by neuroscientist Frédérique Liégeois and her colleagues at University College London. As Liégeois scanned their brains with a functional magnetic resonance imager, the family members listened to nouns and thought of verbs to go with them. The family members with the defective version of *FOXP2* had less activity in a region called Broca's area (FIGURE 14.22). Broca's area, it turns out, plays a pivotal role in processing language (Liégeois et al. 2003).



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FIGURE 14.22

To investigate the role of *FOXP2* in language, neuroscientists scanned the brains of people with defective versions of the gene and their relatives who have working versions. The scans picked up areas of the brain that became active when the subjects listened to words and then thought of other words. In the brains of the subjects with a working version of *FOXP2*, Broca's area became especially active. (It's the large orange spot on the left side of the unaffected brain shown here.) In the brains of affected people, on the other hand, Broca's area was much less active than normal, while other parts of the brain became more active. (Information from <u>Liégeois et al. 2003</u>.)

As we saw in <u>Chapter 7</u>, the sequence of the *FOXP2* gene in humans is significantly different from that of other species. Two more intriguing pieces of evidence about the gene comes from the genomes of Neanderthals and Denisovans. The version of *FOXP2* in both of those extinct hominins was identical to our own. In other words, whatever evolutionary change occurred in the hominin lineage had already taken place in the common ancestor of humans, Neanderthals, and Denisovans 800,000 years ago.

Many language experts doubt that hominins had full-blown language that far back. It's only much later that scientists find evidence of complex behavior that would seem to accompany language—making figurative art and paintings, for example, or trading tools across long distances. The early evolution of *FOXP2* actually may have had little to do with language. After all, *FOXP2* has a number of effects on different parts of the brain, and its evolution could have affected another of those functions.

What makes *FOXP2* all the more puzzling is that the gene shows signs of having undergone strong natural selection in the past 200,000 years. That is an odd sort of timing, since the protein-coding region of the gene had already evolved into its current sequence 800,000 years ago. But as we saw in Chapter 7, the protein-coding region of a gene is not the only kind of DNA that can experience natural selection. Mutations can also change the regulatory regions that determine when and where a gene is expressed—recruiting it for new functions.

To test this possibility, Pääbo and his colleagues returned to the cave at El Sidrón. From a fragment of Neanderthal bone found in the cave, they extracted *FOXP2* along with the DNA surrounding it. Next, they compared this DNA to the same region in living humans.

The scientists discovered an important difference between human DNA and Neanderthal DNA. Humans acquired a mutation in a regulatory region that controls *FOXP2* (Maricic et al. 2012). It's possible that this mutation brought about an important change in the evolution of language. With its new regulation, *FOXP2* was strongly favored by natural selection and swept through our early species.

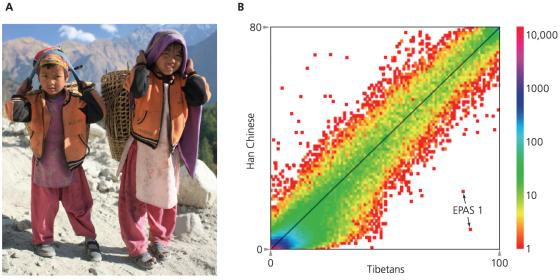
It would be wrong to call *FOXP2* "the language gene," however. Scientists are beginning to uncover other genes that are important to language, and they can now start to probe their evolution. *FOXP2* just happens to have a good head start.

Evolution Meets Civilization

By 40,000 years ago, *Homo sapiens* had expanded throughout Africa and had moved out across the Old World, from Europe to Australia. They were capable of using complex language and crafting sophisticated tools. The brains of our ancestors were powerful enough to create cultures and invent new kinds of technology (Mesoudi, Whiten, and Laland 2006). After 10,000 years ago, humans created a new way to find food, by domesticating animals and plants and inventing agriculture. They used their flexible brains to learn how to turn plants and animals into reliable sources of food, and then they discovered new ways to harvest more food from them. This new knowledge then spread rapidly from one culture to another. In places like Egypt, China, and Mexico, farming opened the way to further cultural evolution: people began to build cities and established nation-states.

Human culture now accelerated, far outstripping the pace of biological evolution. But it would be a mistake to say that human evolution ground to a halt (<u>Stearns et al. 2010</u>). As humans spread across the world, for example, some populations moved into high-altitude regions, where they've had to contend with low levels of oxygen. Several thousand years ago, the ancestors of Tibetans first settled the Tibetan plateau, a vast expanse of steppes that sits 15,000 feet above sea level.

Rasmus Nielsen, a population geneticist at the University of California, Berkeley, and his colleagues wondered if Tibetans had experienced natural selection for adaptations to high altitudes. The Tibetans and the Han people of China are closely related, having descended from a common ancestral population. Ninety percent of Tibetans, Nielsen and his colleagues found, carried two mutations in a gene called *EPAS1* (FIGURE 14.23). The mutations were almost nonexistent in the Han. It turns out that *EPAS1* encodes a protein that helps sense oxygen levels (Yi et al. 2010). Nielsen and his colleagues argue that Tibetans with such mutations were better able to survive at high altitudes and were able to pass on their mutations to more children.



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A: Tibetans have experienced natural selection for living at high elevations. B: Scientists compared genetic variation in a sample of Han Chinese and Tibetan populations. Most alleles occurred at similar frequencies in the two populations (diagonal line). Two alleles that are significantly more common in Tibetans than in the Han occur in the *EPAS1* gene (arrows). This gene is involved in acclimation to high altitude. These high frequencies suggest a history of directional selection on this gene associated with the move to extreme altitude. (Information from Yi et al. 2010.)

Even as humans have adapted to their environments, they've also changed those environments. And in the process, they've opened up new opportunities for natural selection. In <u>Chapter 6</u>, we saw how the domestication of cattle 10,000 years ago created a new selection pressure for lactase persistence. Before the dawn of agriculture, a mutation that allowed humans to drink milk as adults offered no benefit. That's because there was no source of milk, cheese, or yogurt in their environment. But once people began domesticating cattle, they began to live in a milk-rich environment. Once the environment changed, the fitness of lactase persistence alleles rose, and they spread rapidly.

In just the past two centuries, human cultural evolution has wreaked profound changes on our environment. Much of the world has gained access

to clean water, new kinds of medicine and vaccines, and a more reliable supply of food. As a result, rates of mortality have dropped, especially among children. In Germany, for example, 270 in 1000 children died in infancy in 1880. Today less than 4 in 1000 die. The average human height has increased in many countries, but not due to natural selection on certain height alleles. Instead, the increase is an example of human phenotypic plasticity (Chapter 7). An increased supply of food has reprogrammed human growth rates to higher and higher levels with each generation.

In some ways, modern life has reduced the opportunities for natural selection to act on us. An allele that made a child more likely to die from an infection a hundred years ago has much less effect on fitness today, thanks to antibiotics. Even seemingly minor inventions like eyeglasses may be having an effect on human evolution. People who might have once been considered practically blind can today have nearly perfect vision.

Nevertheless, natural selection continues to act on certain traits, even in the most affluent societies on Earth. Stephen Stearns, an evolutionary biologist at Yale, and his colleagues recently studied natural selection in 2238 U.S. women. The women were the subject of a major medical study that has tracked the health of thousands of people in the town of Framingham, Massachusetts, since 1948. The scientists searched for traits that were correlated with having a higher number of children. Then they checked to see whether those traits tended to be passed down from mother to child—in other words, whether they were heritable (Byars et al. 2010).

The scientists discovered that a handful of traits are indeed being favored by natural selection. Women with a genetic tendency for low cholesterol, for example, had more children on average than women with high cholesterol. A greater body weight also leads to greater reproductive success, along with shorter height, lower blood pressure, an older age at menopause, and having one's first child at an earlier age. Stearns and his colleagues have yet to determine exactly what advantage each of these traits confers—a situation that evolutionary biologists often face when documenting natural selection in action. Nevertheless, based on the strength of this natural selection, the scientists predict that 10 generations from now, the women of Framingham will first give birth, on average, a few months younger than those today, will have 3.6 percent lower cholesterol, and will be 1.3 percent shorter.

Emotions and Other Evolutionary Legacies

So far, we've spent this chapter following the evolution of new traits in the human lineage, from bipedalism to sophisticated tools to language to molecular adaptations to modern life. But these are modifications of an ancient body plan. Our ancestors evolved left–right symmetry to their bodies when they were worm-shaped animals crawling on the seafloor 600 million years ago. Despite all the changes that have occurred over the past 6 million years of hominin evolution, we have not broken out of that mold. As Darwin himself wrote, "Man still bears in his body the indelible stamp of his lowly origin" (Darwin 1871).

Our evolutionary legacy also extends into our minds. While we may be unique in the animal kingdom for our abilities to reason, plan ahead, and use language, our behavior still shares profound similarities with that of other animals. Our emotions, for example, bear many hallmarks of their great age (LeDoux 2012).

By 100 million years ago, the basic systems that are essential for many of our feelings had already evolved. Fear, for example, triggers responses from an almond-shaped region on the underside of the brain called the amygdala. The amygdala becomes active at the sight of fearful things, such as a picture of a gun or an angry face. In fact, neuroscientists can observe activity in the amygdala when people see these pictures for just a tenth of a second, which is not enough time for them to become aware they've seen anything. Our brains have shortcuts that can relay information from our eyes and ears to the amygdala without passing through the cerebral cortex. The amygdala, in turn, sends signals to other parts of the brain that produce changes in the body, such as a rapid heartbeat and heightened attention (LeDoux 2007).

Some of the earliest insights about the human amygdala came from studies on the brains of mice and rats. In the mid-1900s, a number of scientists began to experiment on rodents to understand the biology of fear. Like all other mammals, rats and mice have amygdalae, which are linked to other parts of

the brain in much the same arrangement as in the human brain. By implanting electrodes in the brains of the animals, scientists could observe how rodents learned fear as synapses in the amygdala became stronger.

Our fear is similar to the fear experienced by a rat, but not identical. Reading threatening words, like *poison* or *danger*, is enough for the human amygdala to become active. Obviously, that's an experience a rat will never have. But our reactions to those words evolved from the same underlying circuitry we share with our rodent cousins—reactions that likely existed in our common mammalian ancestors. This capacity to feel fear allowed them to respond quickly to threats. If they saw a predator about to attack suddenly, for example, those early mammals could freeze, flee, or retaliate. Some of the dangers that mammals faced were reliable enough that natural selection could produce instinctive fears. Some studies suggest, for example, that we are born with an innate fear of snakes, which would have threatened our primate ancestors for millions of years. But mammals did not have to rely only on hardwired fears. They could also learn a healthy fear for any new dangers they might encounter.

There's more to life than being scared, though. Motivations help mammals reach important goals such as finding food or mates. The most important region for generating these motivations is a small cluster of neurons in the brain stem. If a rat, for example, should be searching for food and unexpectedly get a whiff of something delicious, those neurons will release a tiny surge of a neurotransmitter called dopamine. The dopamine-producing neurons have a vast number of connections to many networks in the brain, and so they can quickly alter how the entire brain functions. Dopamine arouses an animal's attention and also makes it easier for neurons to form stronger connections with other neurons. A rat's brain can begin to associate cues like odors with its long-term goals, such as finding food.

The power of dopamine over the mammalian brain is astonishing. One way to demonstrate its importance is by genetically engineering mice so that they can't produce it. These dopamine-free mice are in many ways perfectly normal. They still prefer the taste of sucrose over other foods, and they can learn where food is located. But they lose the motivation to pursue any goals. They will simply starve from that lack of motivation less than a month after they're born. If scientists give these mice injections of dopamine, however,

they will feed for about 10 hours, until the motivation disappears again (Palmiter 2008).

Too much dopamine can be just as dangerous as too little. Scientists often reward rats by giving them food if they press a lever in response to the right signal—in response to a green light but not a red one, for example. The rat's brain produces surges of dopamine as it learns the rule. If the scientists give the rats an injection of dopamine each time they press the lever, however, something else happens. The rats will keep pressing the lever again and again. They will do nothing else, not even eat. Ultimately, they may die of starvation.

Humans have inherited the same dopamine delivery system. It doesn't make us feel happy so much as eager with anticipation. The rewards that can trigger a release of dopamine are, like our fears, more sophisticated than those that occur in a rat's brain. It can be triggered by winning at a slot machine, the lunge of a fish to a lure, the sight of an attractive face, or even hearing a joke. Unfortunately, the reward system can also be hijacked by substances that cause the brain to release unnaturally large amounts of dopamine. Cocaine and other drugs do just this, and it's the reason that they can become so addictive (Volkow et al. 2010).

Our relationships with other people—particularly with our family—lead to many of our most intense emotions. These bonds also have an ancient history. Among early mammals, strong bonds evolved between mothers and their offspring. Instead of laying eggs and then abandoning them, as reptiles typically do, mammal mothers nurse their offspring. Their young may remain helpless for weeks, months, or even years, during which time they need their mothers' protection.

All living mammals—humans included—use the same hormone to foster mothering (<u>Donaldson and Young 2008</u>; <u>Saltzman and Maestripieri 2010</u>). A gland in the brain known as the hypothalamus releases a hormone called oxytocin late in pregnancy. Some of these oxytocin molecules latch onto receptors in the mammary glands, causing them to begin producing milk. Some oxytocin molecules latch onto neurons in the brain, altering a mother's behavior. In sheep, for example, oxytocin causes ewes to bond with their lambs just after birth. They will be able to recognize the smell and bleat of their own lambs for the weeks that they spend nursing. If scientists block the uptake of oxytocin, however, ewes reject their lambs. If ewes that aren't even

pregnant get an injection of oxytocin, they start behaving like a mother to an unrelated lamb.

Oxytocin is also released by the brains of women both during pregnancy and after birth. The touch of a baby during nursing is enough to trigger an increase of the hormone. Oxytocin tends to cause women to bond more with their babies, as measured by the sounds they make, the number of times they check in on the children, and how much they gaze at them. But experiments in recent years suggest that oxytocin shapes our dealings outside the family as well. Scientists have fashioned oxytocin sprays that can deliver the hormone into the nose. It enters the blood and then eventually reaches the brain, where it reduces the activity of the amygdala. People given oxytocin become more trusting of others and more willing to forgive. They even do a better job of empathizing with other people simply by looking at their facial expressions.

What's striking about these results is not just that oxytocin seems to have taken on many new social roles in our species but also that it is stimulated by different mechanisms. For sheep, rats, and most other mammals, oxytocin is triggered mainly by smell. That's not the case in humans; the release of oxytocin into the bloodstream, and other emotion-related responses, depends much more on our sense of sight. Yet despite this shift, we continue to rely on the same brain structures and molecules for our emotional responses that our ancestors relied on for over 100 million years.

Homo economicus

We humans have powers of thought unprecedented in the history of life. Yet we also make remarkably stupid decisions. These decisions are not just rare flukes of otherwise exquisitely rational minds: we systematically make certain errors in decision making, and even if we're corrected, we go back to making those mistakes again. These mistakes, scientists are realizing, are built into our biology.

The discovery of these built-in irrationalities has had a huge effect on the discipline of economics. Traditionally, economists treated people as perfectly rational agents, always optimizing their finances by making choices that earned them the most money possible in the long term, while minimizing their losses. They liked to refer to this model of the human mind as *Homo economicus*.

Now economists are realizing that *Homo economicus* was a mythical creature. Consider, for example, a simple experiment. An economist hands you a thousand dollars and offers you two choices. The first choice is to gamble. The economist flips a coin, and if it comes up heads, you get another \$1,000. If it comes up tails, you get nothing extra. The second choice is just to get another \$500 with no coin flipping required.

Mathematically speaking, the choices are the same. The people who play it safe get \$1,500; but, on average, the people who gamble also get \$1,500. Yet most people prefer to take the safe \$500 rather than gamble to get more.

Now imagine a twist on the experiment: you start with \$2,000 instead of \$1,000. The choices are the same, but they are choices of loss rather than gain. If you'd like to gamble, the economist will flip a coin; heads means you lose \$1,000, and tails means you lose nothing. Or you can just hand over \$500. The outcomes are the same, and yet people's choices are drastically different from those in the first experiment. People are much more willing to gamble when they are facing a loss than a gain (<u>Dreher 2007</u>).

These experiments show that we are loss averse, so scared of losing money that we will take more risks to hold on to what we have. Loss aversion explains why stockbrokers hold onto stocks when they should sell

—because they hope that the value of the stocks will come back up. It also explains why people are so unwilling to sell their houses at a loss, even when they'd be financially better off without them.

To understand these kinds of bad decisions, economists are starting to collaborate with neuroscientists to better understand how our brains respond to different decisions. Our brains are not all-purpose computers, but decision-making systems that have been shaped by natural selection for hundreds of millions of years. Any organism that can carry out a range of behaviors will regularly face choices between them. Honeybee swarms, for example, must find a new tree cavity to build a hive. They send out scouts that measure up to 20 different cavities at once and report back. Through a kind of honeybee democracy, they settle on one cavity—which, remarkably, is usually the cavity that's closest to the ideal size, shape, and height to ensure their survival (Seeley 2011).

Our mammalian ancestors also made decisions about their behavior. Genetically based decision-making strategies that boosted their chances of survival and reproduction were favored by natural selection over those that lowered fitness. While a mammal like a squirrel cannot use its brain to consciously understand the costs and benefits of different behaviors, its brain can, nevertheless, weigh the squirrel's choices. And its brain networks—the dopamine-producing reward pathway, for example—can produce motivations that cause it to choose action that raises its fitness on average. But our ancestors didn't make decisions in the abstract. They faced ecological choices based on their particular natural history.

Laurie Santos, a psychologist at Yale University, has been investigating the evolutionary roots of our poor economic decisions. To do so, she created a marketplace for capuchin monkeys (<u>Chen, Lakshminarayanan, and Santos 2006</u>; <u>Santos and Hughes 2009</u>). The monkeys could go into a special enclosure in her lab, where they received coins (<u>FIGURE 14.24</u>). A pair of students would then stand in front of the monkeys, each holding a piece of food. In some cases, one student might hold a grape while the other held an apple. The monkeys could put their hand through a hole in the wall and give one coin to one of the students, who handed over food in exchange.



Laurie Santos.

FIGURE 14.24

Monkeys can learn to exchange coins for food. They show the same biases when making economic decisions as people do.

Once the monkeys learned the workings of the marketplace, something interesting happened: they started to behave a lot like humans. They preferred to spend their coin in return for more fruit. They were willing to pay more for more desirable food than less tasty items. They even stole coins from each other—and from the humans.

Then Santos ran a version of the loss-aversion experiment on the monkeys. The monkeys were presented with two students, each holding out one grape. If the monkeys gave their coins to one student, the student always added an extra grape to the first, so that the monkey ended up with two. The other student was less predictable, giving the monkey either just one grape or

three. The choice is the same as the \$1,000 offer for humans. And, like humans, monkeys prefer the safe option. They go for the guaranteed pair of grapes just as often as we go for the safe \$1,500.

Santos then set up a monkey version of the losing choice. The students offered the monkeys three grapes. One student always took one away, giving the monkey two grapes; the other student sometimes took away two grapes and sometimes took away no grapes. And, again, like humans, the monkeys go for the riskier option. They are, like us, loss averse to the point of irrationality.

Since humans and capuchin monkeys have a common ancestor that lived about 35 million years ago, it seems our irrational loss aversion has lingered that long as well. Even when we watch our stock prices plummet, we can't stop ourselves from seeing them the way our primate ancestors perceived losses long ago.

Pleistocene Psychology

Our decision-making networks influence not only our economic choices. They also influence many of the most intimate parts of our lives—including who we find attractive and how parents care for their children.

In some species, animals have a strong preference to mate with individuals that are genetically distinct from themselves. In other words, they are more attracted to individuals with whom they share few alleles. Such a preference may lead to fitter offspring. As we saw in Chapter 6, inbreeding can lead to lower fitness due to homozygosity. Genetic diversity is particularly important in fighting against disease. Vertebrate immune systems recognize invading pathogens with the help of proteins encoded by a set of genes called the major histocompatibility complex (MHC). MHC proteins grab molecules from invading pathogens and display them on the surface of cells. Immune cells can latch onto these antigens and launch a response to fight the infection. Each MHC allele recognizes a limited range of pathogens. If an animal inherits diverse MHC alleles from its parents, it may be able to fight a wider range of diseases. Scientists have documented a preference for MHC-dissimilar mates in many animals, including lizards, fishes, birds, and mice (Havlicek and Roberts 2009; Lie, Simmons, and Rhodes 2010).

Leigh Simmons, an evolutionary biologist at the University of Western Australia, and his colleagues have investigated whether MHC drives mate choice in humans as well. In one study, they showed women 160 pictures of men and asked them to rate the men's attractiveness. They found that the more heterozygous the men were for MHC genes, the more attractive they looked to women. Based on this finding, the researchers suggest that MHC heterozygosity is not only attractive, but that people can reliably detect it from looking at faces (Lie, Rhodes, and Simmons 2008). Simmons and his colleagues also found that women with high MHC genetic diversity tend to have sex for the first time at a younger age and to have more sexual partners, which would also be expected if MHC played a role in sexual selection (Lie et al. 2010).

People can reliably detect MHC diversity by smell as well as from faces. In one of the classic "smelly T-shirt" studies, Claus Wedekind and colleagues at the University of Bern, Switzerland, asked men to wear the same shirt at night for two consecutive nights, and to avoid strong-smelling soaps, deodorants, or lotions over this same period. Women were then asked to rank the shirts according to how attractive they found the male smell to be. Women preferred the smells of men with dissimilar MHC alleles compared to those of males with alleles similar to themselves (Wedekind et al. 1995). What's remarkable in both studies is that the women tested had no idea what MHC genes were, or why they should care about the genotype of potential mates at this locus. They simply scored the phenotypes that were most preferable to them. The underlying predispositions, however, may be there today because of the immunity-related fitness benefits that they accrued to our ancestors.

As we saw in <u>Chapter 9</u>, an animal's fitness depends not just on its success in attracting mates; it also depends on how many of its offspring survive to maturity. In many species, parents invest energy in rearing offspring, but they use a conditional strategy to determine how much to invest. In conditions in which offspring survival is low, for example, parents may abandon offspring. In extreme cases, they may even cannibalize some of their young. When male lions take over a pride, they will sometimes kill the cubs of other males. Unable to nurse their cubs, the lionesses of the pride go into estrus again and can bear cubs for the new males (<u>Hausfater and Hrdy 2008</u>).

Humans show signs of having evolved their own conditional strategies for parental investment. Only an estimated 22 percent of conceptions are carried to term, and the rest end in miscarriages. Many of these miscarriages go unnoticed because they tend to occur early in pregnancy. They can't be considered aberrations since they happen at such a high rate. Instead, there may be an evolved "quality control system" that triggers miscarriages of embryos with abnormalities. As devastating as miscarriages can be for parents, there's good reason to believe that miscarriages are adaptive responses (Boyd and Silk 2009).

Human children are extremely dependent on their parents compared to other animals, thanks in part to the slow development of their brains. Our brains consume an enormous number of calories, and children simply can't get enough of them by gathering food. Our ancestors got their food by either hunting or gathering plants, and it likely took the combined efforts of both parents to keep their children well fed. The strong bonds that parents feel toward their children motivate them to provide for them and to defend them from harm.

But when parents remarry, their children no longer have a biological link to their stepparents. Martin Daly and Margo Wilson, two psychologists at McMaster University in Canada, found that children were seven times more likely to suffer violence from stepparents than from biological parents—a finding that has been replicated in other countries (<u>Daly and Wilson 1988</u>).

Such stark findings do not mean that stepfamilies are doomed to violence. In fact, as the graph in FIGURE 14.25 demonstrates, the vast majority of children in stepfamilies experience no violence at all. And thanks to our flexible brains, we can use knowledge of our evolution to our advantage. By understanding the risks that are a part of our evolutionary legacy, we can find ways to minimize them. This rule holds true not just for our psychological legacy, but our physical legacy as well. By understanding our own evolutionary history, researchers are trying to improve our medical defenses. The topic of evolutionary medicine will be the subject of the next, and final, chapter.

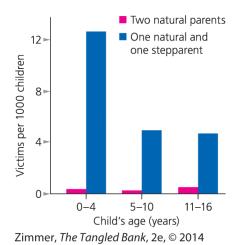


FIGURE 14.25

While child abuse is rare in all families, it is relatively more common in stepfamilies than in families with two natural parents. Studies on the evolution of human parenting can help shed light on such patterns. (Information from Alcock 2009.)

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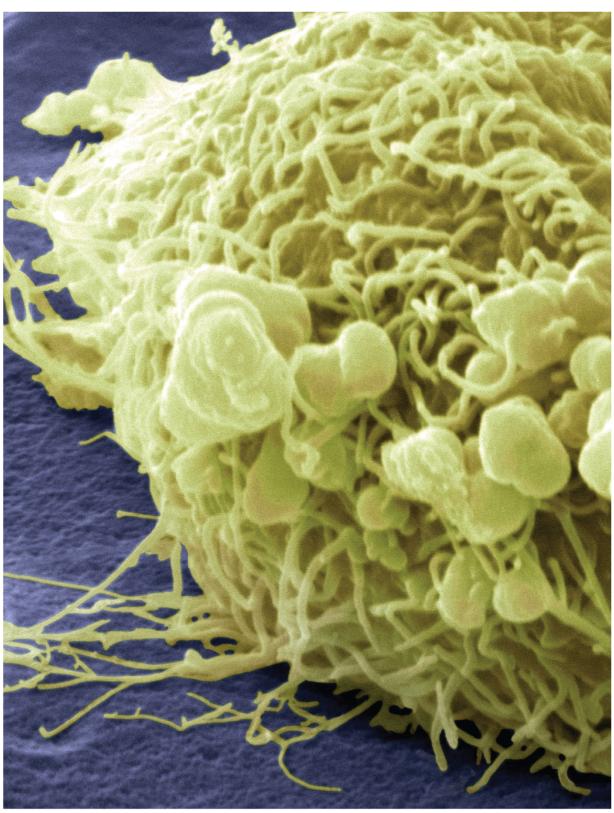
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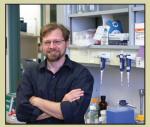
Steve Gschmeissner/Science Source.

Cancer tumors grow and spread through an internal version of natural selection. Mutations enable them to promote the growth of blood vessels that supply them with extra nutrients. Studying the evolution of the genes that build blood vessels is speeding up the search for drugs that can block their growth and potentially starve tumors to death.

Ed Marcotte is searching for new ways to fight cancer. He wants to attack its Achilles' heel—the huge demand that cancer cells have for nutrients. To grow quickly, tumors need extra fuel, which blood vessels supply them with. The tumors themselves send out signals that stimulate new blood vessels to form. Marcotte, a University of Texas biologist, is investigating how to block the growth of blood vessels and thereby starve the tumors.

Scientists know some things about how blood vessels grow, but many mysteries remain. They have identified only a fraction of the genes essential for the process, for example. So Marcotte and his colleagues decided to look for some of the unknown genes. They could then test out drugs that interfered with those genes—and thus block the formation of new blood vessels around cancer cells.

And so the scientists went hunting for blood vessel genes. They didn't look in the human genome, however. Nor did they look in the genome of a mouse or another mammal. Strangely enough, they went looking in yeast—a single-celled fungus (McGary et al. 2010).



Taejoon Kwon.

FIGURE 15.1

Edward Marcotte of the University of Texas studies yeast, plants, and other organisms to find clues about medically relevant genes in humans. His work is one of many examples of evolutionary medicine.

Their choice of yeast was informed by an understanding of evolution. Humans and yeast descend from a single-celled eukaryote that lived over a billion years ago. By comparing them to each other—and to other descendants of that common ancestor—scientists have been able to infer some things about what that common ancestor was like. It already had mitochondria for generating ATP for fuel, for example. It already kept its DNA in a nucleus. It also had networks of genes cooperating to carry out certain tasks. Some gene networks broke down food together. Others eliminated waste. Others relayed signals from the outside environment into the cell's interior.

We saw in <u>Chapter 8</u> how gene networks can remain intact for hundreds of millions of years. The *Hox* gene network that shaped your own anatomy is still strikingly similar to the network that builds the head-to-tail anatomy in a cockroach, for example. We also saw in <u>Chapter 8</u> how these networks can be co-opted for new functions. Long after it first evolved, for example, the *Hox* gene network became co-opted for establishing the coordinates in tetrapod limbs.

Based on studies like these, Marcotte and his colleagues came up with a hypothesis: gene networks involved in blood vessel formation in humans are related to gene networks in yeast, where they carry out some other function. If that were true, it would open up an exciting new way to search for human genes involved in blood vessel formation—and thus to find potential targets for cancer drugs.

The reason for their excitement has to do with how scientists investigate the function of genes. One of the most common ways to do that is to shut down a gene and then observe how an organism's phenotype is affected. It's fairly easy to do this sort of experiment on yeast—partly because yeast cells grow so quickly, partly because you can raise millions of them in a single flask, and partly because scientists have discovered reliable ways to manipulate their genes.

No scientist would do these experiments on humans, however. The closest they will come to such a study is investigating the mutations that give rise to genetic disorders like hemophilia. Many scientists shut down genes in mice to see how they're affected, because the biology of a mouse is strikingly similar to that of humans. But mice are far bigger than yeast and far slower at reproducing, and so insights from these experiments emerge in a comparative trickle.

Marcotte and his colleagues reasoned that if they looked at the scientific literature, they'd find that gene networks in yeast would be far better mapped than the related networks in humans or mice. The scientists might be able to use the well-documented yeast networks to fill in some of the gaps in human gene networks.

To take advantage of this connection, the scientists drew up a list of human genes belonging to the blood vessel development network. Then they searched for their counterparts in yeast. They found a cluster of corresponding yeast genes that happened to belong to the same gene network—a network, it turned out, that repairs the yeast cell wall.

It turned out that scientists had found a number of other genes in the yeast repair network. The scientists were able to find related versions of these genes in the human genome. Many of these genes were still a mystery, their function yet undiscovered. The scientists hypothesized that many of the human genes would also belong to the blood vessel network. And when they carried out tests on frogs and other vertebrates, they discovered that this was, in fact, true. They've now identified eight genes so far that help build blood vessels. In other words, they've found eight new targets for potential cancer drugs.

Marcotte's research (which we'll explore in more detail at the end of this chapter) is part of a growing trend in medical research. Understanding our evolution can yield important insights into the nature of disease and health. In this chapter, we'll explore the fertile intersection of evolution and medicine. Insights from evolution can lead to concrete changes in how doctors practice medicine. These insights also offer deeper lessons about what it means to be human. Natural selection may be able to shape complex adaptations, but it has not made our bodies perfect. We are still left vulnerable to many disorders. In some cases, evolution has actually made us more likely to get sick, not less. In other words, evolutionary medicine helps us understand our maladaptations as well as our adaptations. And finally, some of the most surprising clues medical researchers can hope to find are lurking hundreds of millions of years in the past.

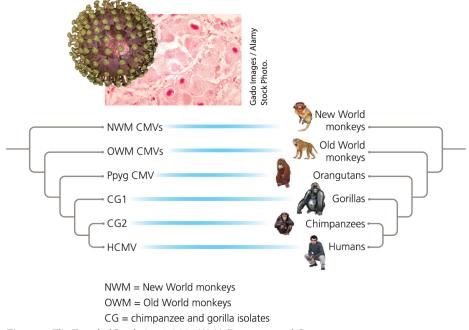
How Diseases Are Born

Every year, 18.3 million people die worldwide from infectious diseases such as malaria, tuberculosis, and influenza (World Health Organization 2004). Each of the pathogens that cause those diseases has its own evolutionary history. Investigating that history can reveal unexpected clues that can help in the fight for better public health.

One way scientists can understand the history of diseases is by reconstructing the phylogeny of the pathogens that cause them. The sort of question they want to ask about a disease will determine the scale of the phylogeny. To make a fine-grained study of an outbreak occurring in a single hospital, scientists might collect samples of the same strain of bacteria from patients and figure out their relationships. To figure out how long ago a pathogen began to specialize on humans, on the other hand, scientists compare it to other species.

To see how scientists can uncover this deep history, consider cytomegalovirus, also known as human herpesvirus 5. Over half of all Americans will be infected with cytomegalovirus by the time they reach the age of 40. In most people, the virus causes no symptoms at all, but for some it is a decidedly less pleasant experience. Cytomegalovirus can cause swollen glands, fevers, and lingering fatigue. Cytomegalovirus is especially dangerous if it infects babies before birth. They may become deaf, blind, or mentally disabled.

Virologists have discovered cytomegalovirus strains in other mammals as well. Each strain is adapted for infecting only a particular species. Human cytomegalovirus cannot infect tree shrews; tree shrew cytomegalovirus cannot infect us. In 2009, Fabian H. Leendertz and his colleagues at the Robert Koch Institute in Berlin constructed a phylogeny of human cytomegalovirus (HCMV) and its closest relatives (FIGURE 15.2). They found that the branching pattern of the viruses mirrored the evolutionary tree of their host species (Leendertz et al. 2009). In other words, the closest relative of human cytomegalovirus, a lineage of viruses called CG2, infects our closest relatives, the chimpanzees.



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FIGURE 15.2

This tree shows how human cytomegalovirus (HCMV, or human herpesvirus 5) is related to other viruses. The relationship of the viruses (left) is a mirror of the relationship of their hosts (right). This pattern suggests that the evolution of hosts and that of their parasites have been locked together for over 30 million years. (Information from Leendertz et al. 2009.)

This mirror-like phylogeny tells us something important about the history of cytomegalovirus: it has been tracking the evolution of its hosts for millions of years. The common ancestor of humans, apes, and monkeys was host to a cytomegalovirus. As that ancestor's descendants diverged into new lineages, the virus diverged as well. It did not leap to distantly related animals, like turtles or sharks. Instead, it continued to adapt to its evolving hosts.

Other pathogens have a different history: they have jumped the species barrier and become human pathogens in recent history. Such is the story of HIV, which started out as a chimpanzee virus and became a human virus in the early 1900s (page 171). It took nearly 20 years for scientists to pinpoint the origins of HIV after its discovery in 1983. Since then, the technology for

isolating viruses and sequencing their DNA has accelerated dramatically, allowing evolutionary biologists to produce phylogenies in far less time.

In November 2002, for example, a mysterious new disease began to spread through China (FIGURE 15.3). At first a Chinese farmer came to a hospital suffering from a high fever and died soon afterward. Other people from the same region of China began to develop the disease as well, but it didn't reach the world's attention until an American businessman flying back from China developed a fever on a flight to Singapore. The flight stopped in Hanoi, where the businessman died. Soon, people were falling ill in countries around the world, although most of the cases turned up in China and Hong Kong. About 10 percent of people who became sick died in a matter of days. The disease was not the flu, not pneumonia, nor any other known disease. It was given a new name: severe acute respiratory syndrome, or SARS.



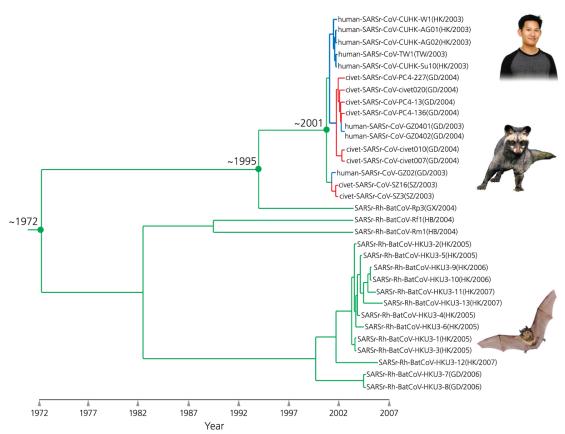
WEDA /Getty Images.

FIGURE 15.3

In 2003, panic swept southeast Asia as a new disease called SARS emerged.

In March 2003, the World Health Organization established a network of labs around the globe to pin down the cause of SARS. Just a month later, they had identified a virus as the culprit. But what kind of virus was it? To answer that question, researchers drew phylogenies based on the virus's RNA. In May 2003, they reported that the SARS virus was most closely related to pathogens called coronaviruses, which can cause colds and stomach flu.

Based on their experience with viruses such as HIV, scientists suspected that SARS had evolved from a virus that infects animals. They began to analyze viruses in the animals that people in China have regular contact with. As they discovered new viruses, they added their branches to the SARS evolutionary tree (FIGURE 15.4).



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FIGURE 15.4

Once the SARS virus was isolated in humans, researchers searched for its closest relatives in animals. As this evolutionary tree shows, the SARS virus evolved from a family of viruses that circulates in bats. The virus then spilled over into palm civets and

humans. Each branch is labeled with a strain code. The color corresponds to the host species. (Information from <u>Lau et al. 2010</u>.)

The first big breakthrough came with the discovery of a closely related virus in catlike mammals called palm civets. The civets are sold in animal markets in China for meat, suggesting a route the virus could have taken from one species to the other. In late 2003, a second SARS outbreak swept China, and scientists traced it to a separate invasion of palm civet viruses into human hosts. But further research revealed that civets are not the main host for the viruses that give rise to SARS in humans. The viruses from both civets and humans belonged to tiny tufts of branches nestled in a large evolutionary tree of coronaviruses that infect Chinese bats (<u>Lau et al. 2010</u>). Viruses from bats regularly infect other animals, which can then pass them to humans.

The Evolution of Host Shifting

Scientists don't have an overarching explanation yet for why some pathogens hop across the species barrier while others remain with their hosts for millions of years. To get closer to such an understanding, some scientists are watching pathogens jump from one kind of host to another in their own laboratories.

At Yale University, for example, Paul Turner and his colleagues study a virus called phi 6, which naturally infects only one host—a species of plant bacteria called *Pseudomonas syringae* (<u>Dennehy et al. 2010</u>; <u>Duffy, Burch</u>, <u>and Turner 2007</u>). The scientists set up experiments to see if phi 6 could switch to a new species of *Pseudomonas*. The different species that make up the genus *Pseudomonas* diverged millions of years ago, so this jump would be equivalent to a baboon-specific virus jumping to a human.

Turner's graduate student Siobain Duffy (now at Rutgers University) prepared dishes containing 14 different species of *Pseudomonas*. She then spiked them all with phi 6 and gave the viruses a day to try to survive on the new hosts. She found that the vast majority of viruses failed.

But a few succeeded. Duffy could see tiny spots in the petri dishes where the viruses were killing bacteria. The viruses in these spots grew far more slowly than they did on their normal host, but they managed to survive nevertheless. Duffy and her colleagues searched the small genomes of the phi 6 viruses to find mutations that had allowed them to cling to existence in their new hosts. They discovered that these viruses all had mutations in the same gene. The gene encodes a protein called P3, which the virus uses to attach to its host.

Such a mutation cannot, on its own, let a virus shift completely from one host species to another. Duffy and her colleagues found that the mutated strains of phi 6 grew very slowly on their new host. What's more, most of the mutations also lowered their fitness on their original host species. This sort of slow growth leaves a strain of viruses at risk of extinction.

It's possible that when moving to a new host, viruses need an intermediate phase that allows them to circulate between old hosts and new ones, replenishing their numbers as new mutations better adapt them to a new host. To investigate this possibility, Turner collaborated with John Dennehy of Queens College in New York. The scientists transferred populations of the virus to flasks containing a different species of *Pseudomonas*. Over the course of 15 days, they periodically added new supplies of the virus from its normal host to the flasks. At first, the scientists found, the virus grew slowly on its new hosts. But over the course of the experiment, its fitness gradually rose in all the flasks.

Turner suspects that the viruses they transferred to the new flasks included a few rare individuals with mutations that just happened to help them survive on the new host. With each transfer, the scientists increased the frequency of beneficial mutations in the virus populations in the flasks. As a result, the fitness of the virus populations steadily increased, allowing them to maintain greater numbers over time. The repeated influx of alleles may have given the pathogen population enough genetic variation to adapt to the selection environment of a new host (<u>Dennehy et al. 2010</u>).

Forever Emerging: The Threat of Influenza

Experiments like Turner's help scientists understand the general principles of evolution that cause new diseases to emerge. Those principles apply not just to viruses that infect bacteria, but to pathogens that pose the biggest public health threats on the planet—and threaten to cause outbreaks in the future that could claim tens of millions of lives.

One of the most worrisome of these pathogens is the influenza virus (FIGURE 15.5). It replicates by infecting the cells lining the airway. The damage it causes can allow bacteria to invade as well, causing pneumonia. Even the response of the immune system can be dangerous, triggering inflammation that makes it hard to breathe. The virus escapes from infected hosts in droplets they cough or sneeze, and it can survive long enough in the environment—floating in the air or stuck to door knobs and other surfaces—to find a new host. Every year, up to half a million people die of the flu worldwide—36,000 in the United States alone.



Photo by courtesy National Museum of Health and Medicine, AFIP (Washington, D.C.).

FIGURE 15.5

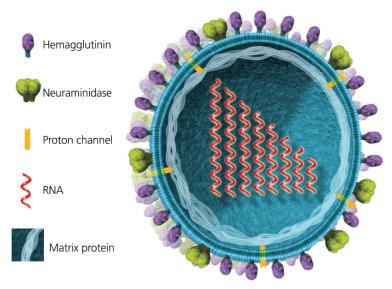
Studying the evolution of influenza is essential to understanding why an outbreak in 1918 was able to kill 50 million people—and to preventing it from happening again.

Each year a combination of flu strains circulates from person to person, and that combination changes from year to year. And every few decades, a distinctly different flu strain appears and often causes far more deaths. The worst of these so-called pandemics occurred in 1918. First appearing in wounded soldiers being shipped home from World War I, it soon spread around the planet. Billions of people got sick, and an estimated 50 million people died.

It's impossible to understand influenza without understanding its evolution. All of the human strains of influenza got their start as bird viruses. In birds, influenza viruses infect the gut rather than the airway, and they typically cause only mild symptoms. Rather than spreading by coughs, the viruses spread in the birds' droppings, which then contaminate water.

Most of these viruses can't successfully infect humans. They use special proteins called hemagglutinin to open up a passageway into cells, and the

shape required to latch onto bird cells often proves a poor fit for human cells (<u>FIGURE 15.6</u>). Even if a bird flu virus can infect a human cell, it takes a long time to multiply, and it may not be able to survive in tiny airborne droplets.



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FIGURE 15.6

The influenza virus contains only 10 genes on eight segments of RNA. It is surrounded by a shell of proteins and a lipid bilayer. The hemagglutinin protein on its surface allows it to enter host cells.

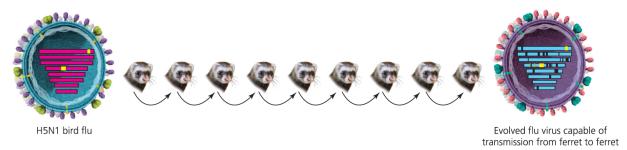
But every now and then, bird flu viruses do evolve to be transmissible in another species. Flu viruses have jumped from birds to humans, as well as to pigs, dogs, horses, and seals. Scientists can document these transitions with phylogenies: they can see how emerging human strains are related to bird strains. Recently, scientists have carried out controversial experiments to see how this transition takes place. They've been studying one of the most worrying strains of bird flu, called H5N1, which has killed over 600 people in Asia and Africa since 2003. Fortunately, it has not yet acquired the ability to spread from one person to the next. How far, scientists would like to know, is the virus from becoming able to spread between humans?

It would be unethical to run such an experiment on humans, so influenza researchers study viruses in ferrets, which show many of the same symptoms

as people when they are infected with human flu strains. In 2012, Ron Fouchier of the Erasmus Medical Center in the Netherlands and his colleagues investigated whether H5N1 could adapt to ferrets. They randomly introduced mutations into the viruses and then sprayed virus-laden droplets into the ferrets (Herfst et al. 2012).

Inside the animals, most of the viruses failed to replicate. A few had mutations that allowed them to invade cells and make new viruses. And those new viruses acquired mutations of their own. (Flu viruses are especially sloppy at copying their genes; just about every new virus has at least one mutation.) Some mutations enabled them to replicate faster than other viruses, and they came to dominate the virus population inside the ferrets.

Fouchier and his colleagues then used the viruses from each ferret to inoculate another ferret, in a method called serial passage. The viruses evolved again inside their new host, and then the scientists repeated the process. After 10 passages, they then tested the virus to see if it had adapted to its mammal host. It had. It could now grow faster in ferrets than at the start of the experiment. And when Fouchier and his colleagues placed a sick ferret next to a healthy one, the virus was able to travel on its own from one mammal host to the next (FIGURE 15.7).



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FIGURE 15.7

Scientists inserted mutations into a bird flu virus and then passed it from ferret to ferret. The virus evolved along the way. When the experiment was finished, the virus had gained the ability to spread between the ferrets by air. (Information from Herfst et al. 2012.)

When the scientists looked at the mutations in the viruses, they discovered just a handful were necessary to allow the pathogen to make the jump to mammals. Some of the mutations altered the proteins that the flu viruses use to latch onto host cells. Others alter how it hijacks the cell to make new viruses (Russell et al. 2012).

Fouchier and his colleagues urge that public health workers monitor H5N1 strains for these mutations, so that we can know in advance if the virus is evolving into a human pathogen. But it's still an open question just how likely it is for those mutations to occur outside of a laboratory. Derek Smith, an evolutionary biologist at Cambridge, and his colleagues created an evolutionary model for the emergence of H5N1 as a human disease based on Fouchier's work. They concluded that it is possible for the virus to acquire all the mutations it needs to make the jump while in a single person (Russell et al. 2012). But while it's possible that this could occur, the likelihood of it happening remains unclear. It might be a one-in-a-thousand event, or a one-in-a-million event.

The Evolution of Virulence

Pathogens don't make us sick and die because they enjoy it. The harm they inflict—what scientists call their virulence—is just a by-product of the way they grow and reproduce. Some pathogens are extremely virulent. Ebola, for example, causes victims to bleed uncontrollably and has a mortality rate of 70 percent. On the other hand, many viruses cause so few symptoms that we're often unaware they're infecting us.

Scientists have found that virulence itself can evolve. One of the most striking examples of this evolution is myxoma virus, which we learned about in <u>Chapter 12</u>. It was introduced into both Australia and France to bring down the populations of rabbits there. In both cases, what started out as an extremely deadly disease evolved into a milder one. Virulence can also increase through evolution, as Kanta Subbarao, a virologist at the National Institute of Allergy and Infectious Diseases, demonstrated in an experiment on SARS (<u>Roberts et al. 2007</u>).

When SARS first emerged, scientists struggled to understand its biology in part because they didn't have lab animals that got sick from the virus the way humans do. Mice could become infected with SARS, but they didn't develop the deadly fevers that made it such a threat to public health. Even when the mice were genetically engineered so that they couldn't develop an immune system, SARS didn't get any nastier.

So Subbarao and her colleagues carried out a serial passage experiment similar to the one Fouchier carried out on ferrets. They infected mice with the SARS virus, gave it a chance to replicate inside them, and then isolated the new viruses to infect new mice. As the virus replicated inside mice and then moved to new hosts, it evolved. Over the course of just 15 passages, it changed from a harmless virus to a fatal one. One sniff of SARS was now enough to kill a mouse.

Why did the myxoma virus and the SARS virus evolve to such different levels of virulence? The answer may lie in the balance between the different kinds of selection that act on pathogens at the same time. All pathogens depend for their long-term reproductive success on two things: their ability

to multiply inside a host and their ability to infect new hosts. Within a single host, the fastest-growing pathogen will outcompete slower ones. But a fast-growing host may undermine its own success by making its host too sick. If its host dies, the pathogen can't find a new host. Even if a host is bedridden, he or she will have less contact with potential new hosts. Thus, when pathogens are going from host to host, selection favors lower virulence.

The result, as we have seen in so many biological situations, is a compromise. These opposing agents of selection together shape the evolution of pathogens, and virulence hangs precariously in the balance. When the population of Australian rabbits was dense, myxoma viruses could move from one host to the next. The most virulent strains reproduced fastest in the rabbits, and they could still spread despite killing their hosts. But in time, the virus killed off so many rabbits that its host population thinned out. Now virulent strains of the virus tended to become extinct because they killed rabbits without being transmitted to another one. Selection began to favor less virulent strains. The result was an unstable balance and intermediate levels of pathogen virulence.

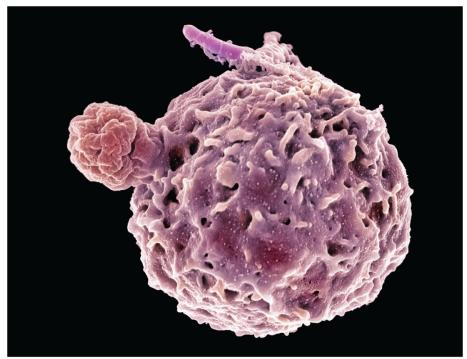
Kanta Subbarao and her colleagues created a virulent strain of SARS by creating the opposite conditions. They passed on the SARS virus from mouse to mouse at the same rate, no matter how quickly it killed its host. Virulence increased very fast once the balancing agent of host-to-host selection was removed (Roberts et al. 2007).

Understanding this balance of selection is critical for public health. Our own actions can make it easier for pathogens to spread, and potentially alter their evolutionary course. We can make it harder for pathogens to spread in many ways, such as washing hands regularly, running vaccine programs, and providing clean drinking water. We change the nature of selection acting on a pathogen in ways that should lead to the evolution of lower virulence.

But we can drive the evolution of virulence in the other way, too, through crowding in prisons or refugee camps, breakdown of hygiene following floods or earthquakes, or even rapid transglobal airline travel that brings infected individuals into contact with a multitude of different host populations. Whenever we make it easy for pathogens to jump from host to host, we tip the tenuous balance in favor of within-host selection, which can lead to rapid evolution of deadly strains with high virulence.

Molded by Parasites

Infectious diseases are, ultimately, the products of coevolution. Pathogens adapt to their hosts, and their hosts, in turn, adapt to the pathogens. Diseases are such a grave risk that all living things have defenses against infections. Even bacteria can recognize invading viruses and chop up their DNA. Animals—and in particular, vertebrates—have evolved particularly elaborate immune systems, made up of many different types of cells that trade signals with one another, swallow up some pathogens, and manufacture chemical weapons to destroy others (FIGURE 15.8).



SPL/Science Source.

FIGURE 15.8

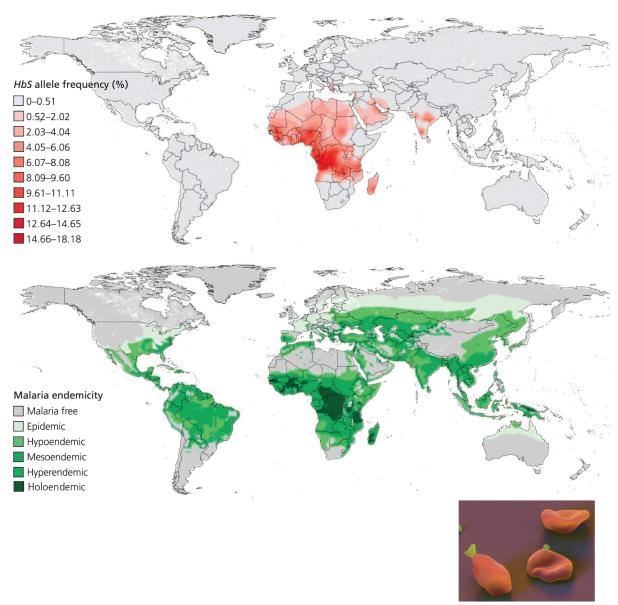
A white blood cell attacks invading bacteria. Mutations that improve its performance are strongly favored by natural selection.

Despite the power and sophistication of the human immune system, our pathogens continue to thrive. The high-speed evolution of pathogens means that natural selection is always favoring new defenses in their hosts. When evolutionary biologists measure the strength of selection on the human genome (page 175), immune system genes consistently turn up near the top of the list (Fumagalli et al. 2011).

In the last chapter, we saw how humans have been shaped by natural selection over the past few thousand years as they've expanded into new habitats and developed sources of food. They've also evolved defenses to diseases during the same period.

Malaria has had a particularly powerful effect on the human genome. The disease appears to have emerged as a serious threat within the last few thousand years in Africa, probably triggered by the spread of agriculture through the continent. As farmers cleared forests, malaria-spreading mosquitoes could breed in the standing water in their fields and then infect the farmers as they slept in their villages. The disease spread wherever mosquitoes could carry it—even as far north as England and the upper reaches of the United States. Malaria is gone from England and the United States and many other temperate countries, thanks to larvicide in standing water and insect-proofing houses with screens. But in Africa and elsewhere, malaria remains a major scourge that infects 250 million people a year and kills 880,000.

Mutations that provide resistance to malaria have been able to spread quickly in regions where the disease is especially common. Sickle-cell anemia (page 137) is the by-product of one of those defenses. People suffer from sickle-cell anemia when they inherit two copies of an allele called *HbS*. If they get one copy, however, they are only one-tenth as likely to get severe malaria. Carrying a single *HbS* allele has thus provided a huge boost in fitness. As a result, human populations where malaria is prevalent show much higher frequencies of the *HbS* allele than other areas where malaria is less common or absent (Piel et al. 2010; FIGURE 15.9).



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FIGURE 15.9

Malaria is caused by a single-celled protozoan that invades blood cells (shown here in green in the inset). The prevalence of malaria (bottom) aligns with the frequency of the *HbS* allele. This pattern supports the hypothesis that the allele protects against malaria. (Information from <u>Piel et al. 2010</u>.)

Darwinian Vaccines

Much of medicine is an enhancement of our natural defenses against diseases. Vaccines, for example, prime our immune systems to recognize pathogens quickly, so that they can mount a defense before the pathogens can take hold (FIGURE 15.10). They have protected billions of people and made once-common diseases rare. Smallpox, a disease that once killed tens of millions of people every year, was eradicated thanks largely to a vaccination campaign in the 1960s and 1970s.



B BOISSONNET / BSIP/Superstock.

FIGURE 15.10

Vaccines for many diseases contain weakened viruses. The viruses are rendered safe through evolution.

Vaccines are also a demonstration of applied evolution (<u>Hanley 2011</u>). The vaccines for many viral diseases contain live viruses, which are too weak to make people sick, but strong enough to trigger a protective immune response. To make these live-virus vaccines, scientists use serial passage.

For the Sabin Type 1 vaccine against polio, for example, they infect a monkey with polio virus. Monkeys are not the natural host for polio, and so the viruses initially do a poor job of replicating. After 24 passages from monkey to monkey, they become better at infecting monkeys—and worse at infecting humans. Polio attacks nerve cells; to make it harder for them to do so, scientists then infect monkey kidney cells in flasks with the viruses for 48 generations. At the end of this procedure, the scientists have evolved polio viruses that are well adapted to infecting monkey kidney cells but have lost their ability to paralyze human victims. Thanks to vaccines such as these, polio is now eradicated from most of the world.

Some diseases have been harder to fight with vaccines. Influenza infects 30 to 60 million people every year, even though flu vaccines were invented over 50 years ago. It's a good idea to get a flu vaccine if your doctor recommends it, but it's important to recognize that it's not a perfect defense. In a typical flu season, flu vaccines may provide only about 60 percent protection. And that protection lasts only for the season—the next year, you'll need to get yet another vaccine.

Evolution is the reason for the limits of flu vaccines. During a flu infection, our immune systems learn to produce antibodies that can latch tightly onto the tip of the hemagglutinin protein, preventing the virus from latching onto host cells. Flu vaccines, which contain killed flu viruses, trigger a similar response. Certain immune cells retain their ability to recognize the same features of the hemagglutinin proteins, so that if live flu viruses invade, they can quickly mount a response.

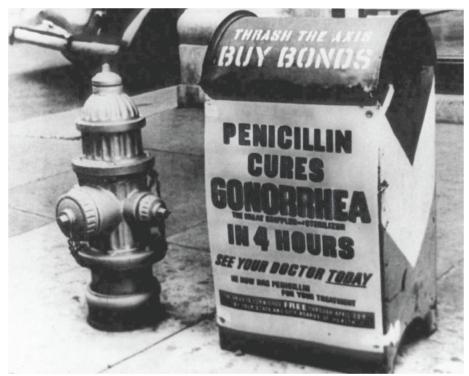
Unfortunately, the hemagglutinin protein can evolve slightly different shapes. These mutants can then avoid the clutches of the body's antibodies and still be able to latch onto host cells. Natural selection strongly favors these mutants, which swiftly take over the virus population. The continual evolution of influenza viruses lets them escape the immune system, and it also makes the protection from vaccines useless after a few months. By then, the flu viruses in circulation will have mutations that require a different kind of immune response.

Some flu researchers are looking for a way to outfox influenza's high-speed evolution. They've engineered antibodies that latch onto other parts of the flu virus. Some of these antibodies provide protection against many different strains of the flu. It's possible that the scientists have discovered

parts of the flu virus that aren't as evolutionarily flexible as the tip of the hemagglutinin proteins. Mutations can allow them to evade antibodies, but they also prevent the virus from replicating. Some day, we may need to get these so-called universal flu shots only a few times in our lives, rather than every year.

Silver Bullets

Along with vaccines, antibiotics have revolutionized the fight against infectious diseases. In the mid-1900s, scientists discovered that fungi and bacteria made compounds that could stop bacterial diseases such as tuberculosis and gonorrhea. Infections that once almost certainly would have been lethal simply disappeared in a matter of days. Some optimists declared that infectious diseases soon would be a thing of the past (FIGURE 15.11).



National Institute of Health.

FIGURE 15.11

When antibiotics were discovered in the mid-1900s, they were hailed as a miracle drug. Since then, bacteria have evolved increasing resistance to them.

But not long after antibiotics first became available, doctors began reporting that they sometimes failed. In the 1950s, Japanese doctors used

antibiotics to battle outbreaks of dysentery caused by *E. coli*, only to watch the bacteria develop resistance to one drug after another.

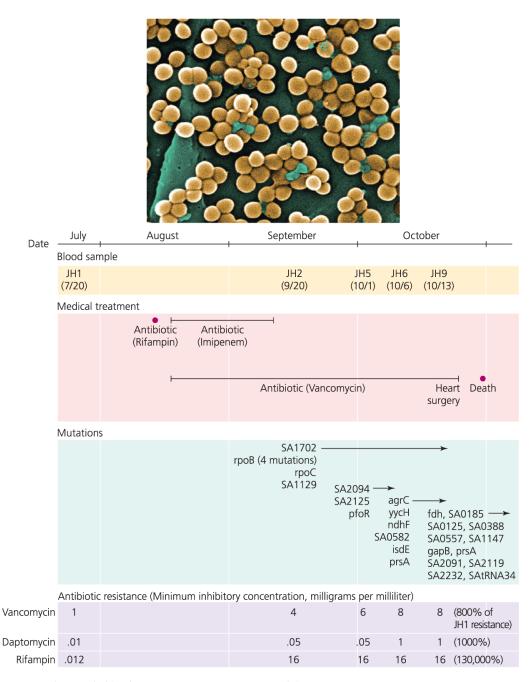
The Japanese doctors had come face to face with one of evolutionary medicine's most sobering lessons: medicine itself can drive the evolution of disease. The microbes that breed in an infection also mutate, and some of those mutations may help the microbes to resist an antibiotic. In the absence of an antibiotic, those mutations may not increase their fitness. In fact, they may even make the microbes grow more slowly. As a result, those mutations will remain rare. But antibiotics can alter the evolutionary landscape in a flash. If a mutation provides a microbe with even a little resistance, it will have more reproductive success than vulnerable microbes that die off altogether.

Resistance can evolve in many ways. Some mutations can make it harder for antibiotics to attack their targets inside the microbe. Some alter membrane pumps so that the microbes can flush the antibiotics out quickly before they cause serious harm. As a lineage of resistant microbes takes over a population, new mutations emerge, some of which can make the microbes even more resistant. The evolutionary costs of these mutations can be eliminated by new mutations, which allow the microbes to reproduce just as quickly as vulnerable strains. These compensatory mutations allow resistant microbes to survive when there are no antibiotics to give them an edge.

Scientists can observe antibiotic resistance as it evolves in laboratory experiments (Perron, Zasloff, and Bell 2006). They're also learning how to observe this evolution within the bodies of infected people. Alexander Tomasz, a micro-biologist at Rockefeller University, and his colleagues have tracked the evolution of resistance in a single patient, known only as JH (Mwangi et al. 2007). In 2000, JH developed an infection of Staphylococcus aureus bacteria in a heart valve. He was treated with an antibiotic called rifampin, which failed to work; his doctors then gave him heavy doses of more powerful antibiotics, such as vancomycin, which failed as well. After three months of treatment, surgeons replaced JH's heart valve, but he died two weeks later.

Tomasz and his colleagues were able to isolate the bacteria from a series of five blood samples that doctors had taken from JH over the course of the infection. They sequenced a *Staphylococcus aureus* genome from the first sample and then analyzed the DNA from later samples. The bacteria from the

later samples shared a set of genetic markers with the original sample. Those markers indicated that the bacteria had evolved within JH's body, rather than having invaded later (FIGURE 15.12).



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In 2000 a patient known as JH developed an infection of *Staphylococcus aureus* (top). Doctors took a series of blood samples and identified the new mutations that arose in the bacteria over the course of the infection. Mutations that conferred more resistance to antibiotics were favored by natural selection. (Information from Mwangi et al. 2007.)

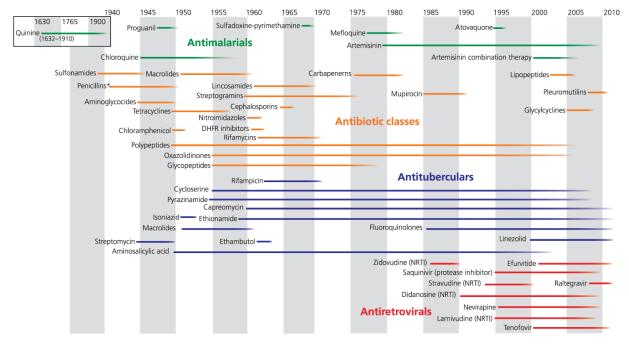
Over the course of the infection, Tomasz and his colleagues found, the bacteria evolved increased resistance to three out of four antibiotics. The bacteria became eight times more resistant to vancomycin, for example, and 1000 times more resistant to rifampin. These changes were the result of mutations that arose in the bacteria and were favored by natural selection. In all, Tomasz and his colleagues pinpointed 35 mutations that distinguished the bacteria in the last sample from those in the first. They could even see the mutations accumulate from one sample to the next. Some of the mutations are familiar to scientists from other resistant strains of bacteria; others are new, altering the bacteria in ways the scientists have yet to understand.

The bacteria in JH built up new mutations through vertical gene transfer. But bacteria can also acquire genes through horizontal gene transfer, which can speed up the evolution of antibiotic resistance dramatically. Many species of bacteria that live in the soil have genes that can, by coincidence, provide resistance to antibiotics; from time to time, they can pass those genes on to bacteria that cause human infections. Once genes evolve high levels of resistance, they can move from one species to another, either in the soil or in our bodies.

Resistance genes may start out being transferred individually, but over time they can be combined. Some of them are carried on ringlets of DNA called plasmids that bacteria exchange. Those plasmids sometimes mutate, splicing together their DNA so that resistance genes from separate plasmids can end up together on one. A plasmid resistant to two antibiotics may raise the fitness of bacteria much more than a plasmid with just one. And if it should pick up a third resistance gene, its fitness rises even more. This is probably what happened in Japan during the dysentery outbreaks: drugs fostered the evolution of *E. coli* resistant to several antibiotics.

Today, new strains of pathogens are emerging that are resistant to just about every antibiotic on the market (<u>FIGURE 15.13</u>). The only way to cope with the crisis is to treat it as an evolutionary phenomenon. It's not enough to

recognize that evolution is taking place; doctors need to understand the complexities of that evolution. Some hospitals have tried to fight resistant bacteria by rotating their antibiotics over the course of a few months, so that the bacteria don't have much time to evolve increased resistance. That strategy hasn't worked, and mathematical models of evolution show why: a more promising strategy, according to the models, is to give different antibiotics to different patients, to slow down the transmission of bacteria between them (Bergstrom, Lo, and Lipsitch 2004).



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FIGURE 15.13

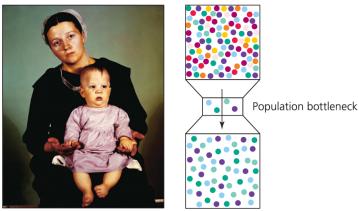
The history of the development of drugs against bacteria and other pathogens is one of ongoing failure. When antibiotics are first introduced, they are overwhelmingly effective. But within a matter of years, strains of resistant bacteria evolve.

Human Variation and Disease

Each of us is born with a unique collection of mutations. For some of us, those mutations are a boon, providing us with long lives. For others, they are a burden, making us vulnerable to heart disease and cancer. And millions of people have the misfortune to carry mutations that cause genetic disorders leading to blindness, debilitation, and death.

By exploring our evolutionary history, we can come to better understand how these variations arise in our species. When people live in small populations, for example, they can become victims of genetic drift, which can make even harmful mutations common. On the remote Pacific island of Pingelap, for example, 5 percent of the population is completely color-blind (Hussels and Morton 1972). By comparison, only 0.003 percent of the U.S. population suffers from this condition, called achromatopsia. Historical research indicates that around 1775, a typhoon reduced Pingelap's already small population to just 20 survivors. One of those survivors carried one copy of the achromatopsia allele. Thanks to that twist of fate, 5 percent of the island's current residents now suffer from the disease because they carry two copies of the original allele, while 30 percent carry a single copy.

Islands are not the only places where genetic drift drives up genetic disorders. In the late 1600s, a small group of closely related farmers traveled from Switzerland to Germany and finally to the United States, where they became known as the Amish. Shunning intermarriage with other groups, they kept to themselves, and they still do today. In effect, they've created a "genetic island" in the middle of a continent. Not surprisingly, the Amish suffer high rates of certain genetic disorders, such as Ellis-van Creveld syndrome, which leads to extra fingers and dwarfism (FIGURE 15.14). Scientists have been able to trace that disorder to a single Amish couple who immigrated to Pennsylvania in 1744.



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FIGURE 15.14

Some human populations suffer from high rates of certain genetic disorders. The Amish, for example, are unusually likely to suffer from Ellis-van Creveld syndrome, which causes deformities to the skeleton. The Amish descend from a small group of immigrants from Europe who settled in the United States. Their genotypes are not a representative sample of the genotypes in their homeland, because the initial population was so small. Because of these types of founder effects, even one person carrying genes for a rare genetic disorder makes it much more common in the population.

Another form of genetic variation that's important for medicine is the one determining how people respond to drugs. Certain alleles can lead to harmful side effects from drugs that are safe in most people. Different alleles can lead some people to require a larger dose of a drug than others in order to get an effective treatment. Scientists still know relatively little about how genes determine drug responses; the more they learn, the better they can personalize the medicine doctors give to their patients (Evans and Relling 2004; Sadée and Dai 2005).

Some clues to drug responses can be found in our evolutionary history. Warfarin, used to destroy blood clots that can cause heart attacks and embolisms, is the most commonly prescribed anticoagulant worldwide. In some people, however, a standard dose of warfarin can lead to lethal uncontrolled bleeding. Yet doctors can find the right dose for patients only by trial and error. Genetic factors account for some of the variance in the

response of patients to the drug. Genes have been found to explain about half of the variance (Ross et al. 2010).

Kendra Ross of the University of Toronto and her colleagues surveyed alleles of warfarin-linked genes in 1279 people representing a number of major human populations worldwide. They found that one of these genes, *VKORC1*, has a striking pattern of geographical variation. People with an allele of the gene, known as the rs9923231 *T* allele, require only a low dose of warfarin and are at greater risk of bleeding. Ross and her colleagues found the allele in 100 percent of Han Chinese but only 10 percent of Africans. Using several different tests, the scientists found strong evidence for natural selection as the cause of the fixation of the rs9923231 *T* allele.

It will be intriguing to learn the cause of selection for this allele. The protein encoded by *VKORC1* is involved in blood clotting as well as other functions such as bone mineralization. Whatever the cause, natural selection has also made many people vulnerable to warfarin. Just as drugs have side effects, natural selection can have side effects of its own.

The Natural History of Cancer

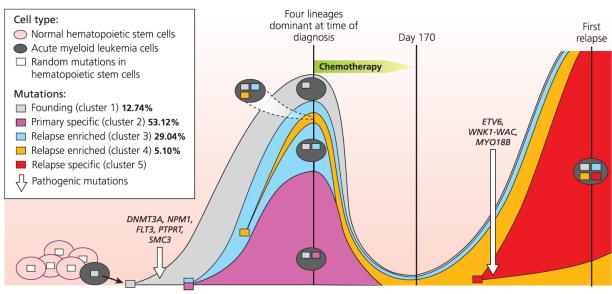
Roughly 900 million years ago, our ancestors evolved from single-celled protozoans into multicellular animals. Instead of reproducing independently, the cells in animals began to cooperate. They developed into different tissues that together produced a working body. The fitness of their genes rose and fell together as they enabled animals to feed and reproduce. The cooperation between cells allowed animals to occupy new niches that their single-celled ancestors could not. But along with these evolutionary benefits, these new bodies presented a new risk. Cooperation provides the opportunity for cheaters to thrive. In our own bodies, this cheating is known as cancer.

Every time a cell divides, there's a tiny chance that a mutation will occur. In some cases, those mutations strike genes that control the rate at which cells divide. These gatekeeper genes, or proto-oncogenes, ensure that cells divide only when they need to, and stop dividing when they shouldn't. For example, when you cut yourself, cells in the skin and other tissues rapidly divide to heal the wound. If they were to keep dividing, they'd create an expanding mass of flesh. Mutations to these gatekeeper genes (mutated versions of these genes are known as oncogenes) allow cells to grow faster than their neighbors. In some cases, these runaway mutant cells can form a tumor.

Within a developing tumor, cells continue to mutate, and cells with mutations that speed up their growth come to dominate the population of cancer cells. Some genes, for example, normally become active only in sperm cells, helping them to grow rapidly throughout a man's adult life. Normally these sperm-growth genes are kept silent in other parts of the body. But mutations can switch them on in cancer cells, making them divide faster.

Natural selection allows cancers not only to become more aggressive but also to become harder to treat. As tumors grow, some mutations may make them resistant to drugs. Much like bacteria, those resistant mutants will be able to grow faster than susceptible cancer cells. Scientists can document this evolution by surveying cells at different stages of a cancer. Rare resistant cells become more common, and new mutations arise and spread. **FIGURE**

<u>15.15</u> shows the evolution that occurred in a single person before and after chemotherapy (<u>Ding et al. 2012</u>).



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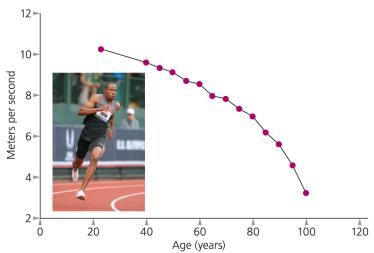
FIGURE 15.15

This diagram illustrates the evolution that occurred in cancer cells in a woman who died of acute myeloid leukemia, a cancer of immune cells. The immune cells arise from progenitors called hematopoietic stem cell (pink circles on the left). One lineage of these cells acquired mutations, including the five indicated on the diagram (*DNMT3A*, *NPM1*, *FLT3*, *PTPRT*, and *SMC3*). Able to grow faster, the cancerous cells became more common, as indicated by the increasing width of the gray wedge. Within this lineage, some cancer cells acquired new mutations, two of which are represented here by the blue, orange, and purple rectangles. The descendants of the cells with these mutations rapidly became more common, thanks to natural selection. When the patient was diagnosed with leukemia, four lineages were dominant. The percentages at diagnosis are indicated in the box. Chemotherapy drastically reduced the population of all these lineages. But cluster 4 cancer cells (orange), which had previously been the rarest of the four lineages, eventually rebounded. New mutations within this lineage (red rectangle) then produced cells that came to dominate the population. The cancer thus evolved resistance to chemotherapy. (Information from Ding et al. 2012).

The same mathematical models that help evolutionary biologists to understand the rise of antibiotic-resistant bacteria are now being adapted to shed light on the evolution of resistance to anticancer drugs in tumors (Merlo et al. 2006). Cancer cells follow the rules of natural selection, so it's important to start treating tumors as early as possible because the longer they evolve, the more likely it is for a resistant mutation to arise spontaneously in a cell.

Why the Fountain of Youth Runs Dry

Medicine has helped to dramatically increase the average life span of people worldwide. A child born in the United States in 1900 could expect, on average, to live for 47 years. A child born in 2010 can expect to live 78 years. But there's actually less to our longer life span than meets the eye. Most of the progress has come from saving the lives of children as well as young mothers who used to die in childbirth. At the other end of life, people today still age in much the same way people did a hundred years ago. Their bones get brittle, they lose their stamina, and they lose their ability to fight infections. The decline begins in middle age and continues gradually for decades (FIGURE 15.16).



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FIGURE 15.16

This graph illustrates how the human body declines with age. Using world-record times for the men's 100-meter run by age groups, scientists can calculate the average speed of each championship runner. The maximum speed starts dropping in the third decade of life. Evolutionary biologists seek to understand why this decline evolved. Andy Lyons/Getty Images, (Information from Stearns and Koella 2008.)

As the world's life span increases, an increasing fraction of the population will be older. In 2012, there were approximately 810 million people aged 60 years or over in the world. By 2050, that number is projected to grow to more than 2 billion. That will mark a milestone in human history: older people will for the first time outnumber the population of children ages 14 and under. Many of them will face the diseases of old age, from arthritis to cataracts to Alzheimer's disease. To cope with this growing medical challenge, scientists are investigating the biology of aging. Understanding our evolution can help explain why we get old, and what we might be able to do about it.

Aging is not unique to humans. As we saw in <u>Chapter 9</u>, other animals have their own typical life spans, at the end of which their bodies decline. That decline is typically the result of a trade-off between natural selection acting during early years and late ones. Many human diseases illustrate this trade-off. Huntington's disease is a devastating genetic disorder that slowly destroys the nervous system. It is much more common than most fatal genetic disorders: about one in 18,000 people suffers from the disease. If the alleles causing Huntington's disease affected people throughout their lives—when they were teenagers, for example—the alleles would be much rarer than that. In reality, most people do not start to suffer its symptoms until their 40s. They have enough time to raise children, passing on the disease-triggering alleles to the next generation before they die.

This trade-off can explain how our bodies have ended up defending us from diseases in a less than perfect way. One of the most important controls against cancer is the p53 tumor suppressor protein. It responds to stress inside cells, particularly to damaged DNA, which may signal the first steps toward cancer. It can cause a cell to die or to stop dividing. In either case, p53 prevents the cell from possibly growing into a tumor, but it takes a toll in the process. As the years pass, p53 can kill or stunt so many cells that tissues can no longer renew themselves. By forcing cells into early retirement, p53 may prevent them from becoming tumors, but the cells may damage surrounding tissue and release abnormal proteins that stimulate the growth of cancer cells.

In other words, p53 is an effective stopgap defense against cancer. It helps keep young people relatively cancer-free. But it also damages the body in the process. The damage accumulates slowly, over the course of many years. By

the time it has an impact on us, we're so old that natural selection cannot alter it.

None of this means that we can't hope to extend the human life span. Some of the most promising results in the study of longevity have come from experiments in which scientists reduce the amount of food animals can eat. If they cut the normal number of calories in an animal's diet, it often lives much longer. Some experiments suggest that restricting an animal's diet triggers a special response in its cells. They begin to produce proteins that can repair the damage caused by the stress of not getting enough to eat.

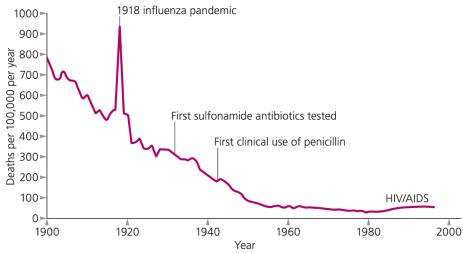
This response appears to be an ancient strategy, given that the same stress-fighting genes can be found in animals ranging from nematode worms to mice. These genes may have evolved as a way to cope with short-term stress, such as famines and droughts. Scientists who study nematode worms have found that they can double the life span of the animals with mutations that keep these genes switched on. The genes may be able to continually repair damage to cells, fighting the effects of aging. Restricting calories may have the same effect, by keeping the genes switched on permanently. It may someday be possible to switch on these genes by taking a pill, rather than by going hungry.

But evolutionary biology offers a warning about antiaging drugs. Nicole Jenkins, a biologist at the Buck Institute for Age Research in California, and her colleagues have found that mutations that bring long life to nematode worms actually lower their fitness (<u>Jenkins et al. 2004</u>). The scientists put 50 of the long-lived worms in a dish with 50 normal worms and then let them breed. Jenkins then randomly picked out 100 of the eggs and used them to rear the next generation. The long-lived worms were just as fertile as the normal worms, Jenkins found, and yet within just a few generations they had vanished from the dish. They had been outcompeted by the shorter-lived worms.

Natural selection, once again, did not favor long life simply for long life's sake. Jenkins's experiment raises the possibility that taking resveratrol or some other medication to prolong aging may bring with it some kinds of side effects. It would be nice to have an elixir of life, but an understanding of evolution's trade-offs shows why that's so unlikely.

Mismatched with Modern Life

For people in the United States and other developed countries, it's nearly impossible to imagine the suffering infectious diseases brought a century ago. Along with tremendous outbreaks of such scourges as influenza, many other diseases also steadily killed off people year in and year out. Since then, the death rate from infectious diseases has dropped dramatically (FIGURE 15.17).



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FIGURE 15.17

The death rate from infectious diseases dropped dramatically in the United States over the twentieth century. It began to decline thanks to better hygiene, clean drinking water, and better food. The invention of antibiotics in the mid-1900s helped push the death rate even lower. The HIV epidemic that began in the late 1980s raised the death rate, although it remains far lower than at the beginning of the century. (Information from Stearns and Koella 2008.)

Yet, in the countries that have experienced these drops in infectious diseases, people have seen an increase in another kind of disease. Rather than being caused by pathogens, these rising diseases are caused by our own immune systems. Crohn's disease, for example, occurs when the immune

system attacks the lining of the intestines. Asthma is caused by inflammation in the lungs. Type 1 diabetes occurs when immune cells attack insulin-producing cells in the pancreas. All these diseases were once rare in the United States and other developed countries but now are increasingly common.

These diseases seemed to have followed in the wake of affluence. They first emerged in countries such as England and the United States, which were among the first nations to go through an industrial revolution and then improve their public health. Later, when other countries went through the same transition, they also saw a rise in autoimmune diseases. Even within countries, a similar pattern can be found. In Venezuela, for example, the population is split mainly between cities and farms, and some Indians still live in isolated villages in the rain forest. Venezuelan city dwellers have higher rates of allergies than Venezuelan farmers do, and Venezuelan Indians have no allergies to speak of.

In 1989, David Strachan, an epidemiologist at the London School of Hygiene and Tropical Medicine, suggested that these autoimmune diseases were breaking out because children had become too clean. They were not being exposed to dirt and dust, and they were not being infected by bacteria and parasitic worms. As a result, their immune systems were attacking themselves, rather than pathogens. Today, scientists refer to this as the hygiene hypothesis (Yazdanbakhsh, Kemsner, and van Ree 2002).

The hygiene hypothesis builds on the fact that life in the twenty-first century is very different from that during our 500-million-year history as vertebrates. Our ancestors were under constant assault from pathogens. They could not respond with antibiotics or bandages. Instead, natural selection responded by fine-tuning the immune system, which became better able to cope with the infections (Rook 2010). A more powerful immune system brought with it new dangers. The weapons it used against pathogens could also damage the body's own tissues. If the antigens it responded to were too diverse, it might also attack beneficial bacteria that helped the body digest food. It might even mistakenly learn to attack the body's own cells as if they were pathogens.

In response to these risks and benefits, a delicate balance evolved within the vertebrate immune system. It can eradicate some pathogens, but it also tolerates many others. As a result, a wild vertebrate can carry a number of pathogenic species, from bacteria to intestinal worms, without triggering a major reaction from the immune system. The immune system apparently must learn this tolerance, however. Mice that are experimentally reared with no bacteria in their bodies will frequently develop autoimmune disorders.

The hygiene hypothesis holds that children in affluent countries are no longer exposed to the typical bacteria and parasites that their ancestors were. Because they play inside houses rather than out in the dirt, many children do not get exposed to as many microbes. When these children get sick from bacteria, they are treated with antibiotics that kill many harmless bacteria along with the pathogenic ones. Intestinal worms are now a thing of the past. All these changes mean that the immune systems of our protected children don't get the correct cues to develop the right level of tolerance. They have become more likely to overreact to a harmless antigen or even attack the child's own body.

Scientists have been testing the hygiene hypothesis from many different directions, and their results are striking. Martin Blaser and Yu Chen, microbiologists at New York University, have been looking at the effects of one particularly important species of bacteria called *Helicobacter pylori*. *H. pylori* lives in the stomach, and it's had an especially intimate relationship with our species. Its phylogeny mirrors our own. *H. pylori* strains that live in Native Americans are more closely related to the ones in Asian people; African strains of *H. pylori* are more diverse than strains on other continents.

H. pylori was ubiquitous in our species before the advent of antibiotics, but it's now on the decline. Only one in five American children now carries it. Blaser and Chen analyzed the medical histories of more than 7400 people who took part in a nutrition survey. As part of the survey, researchers collected stool samples. Blaser and Chen checked the samples for signs of *H. pylori*. In 2008, they reported that children between three and 13 years old who carried *H. pylori* were 59 percent less likely to have asthma than children who were free of the bacteria (<u>Chen and Blaser 2008</u>).

On the whole, antibiotics and better hygiene have made the world a healthier place. But that's small comfort to people who suffer from the autoimmune diseases that may have been produced as a side effect. Some researchers are using the hygiene hypothesis to figure out ways to treat these diseases of modernity. Some doctors have dispensed parasitic worms to people suffering from Crohn's disease, and they've found that the parasites

tend to reduce the symptoms (<u>Elliott and Weinstock 2009</u>). These experiments are just a proof of principle, however. Ideally, doctors would be able to prescribe drugs that would trigger the same response as the worms and bacteria, without the harm that they can cause. (*H. pylori*, for example, may protect children from asthma, but it also increases the chance of developing stomach cancer.) It's possible that someday people will swallow pills containing surface proteins from parasitic worms or bacteria to teach their immune systems how to behave themselves.

The hygiene hypothesis explains just one of many mismatches between our evolved bodies and our modern world. Like other organisms, we have an internal clock that controls how our bodies function at different times of day. It creates 24-hour fluctuations in our appetite, our body temperature, and our wakefulness. When sunlight enters our eyes each day, it resets the clock. Today, however, we are confusing our bodies. We keep lights on well past dark, for example, and we sometimes fly from one time zone to another. Lights at night and long-distance plane travel have both been linked to an increased risk of cancer, possibly because they disrupt our hormone cycles. Modern agriculture has provided much of the world with cheap, abundant calories in the form of sugar and other carbohydrates. Our appetite for such foods evolved when they were far scarcer. Now they are leading to a global epidemic of obesity and diabetes (FIGURE 15.18).



Joanna Pecha/Getty Images.

FIGURE 15.18

Obesity and diabetes have become global threats to public health. Our bodies are poorly adapted to the modern environment, in which sugar and other carbohydrates are available in enormous supplies.

The sudden mismatches between our bodies and modern life have changed the fitness of our alleles. As we've seen in previous chapters, the fitness of a mutation is not some absolute value. It depends on which other genes a mutant gene shares its genome with, and it also depends on the environment in which its owner lives. This perspective changes the way we think about genetic disorders. What is healthy in one century may not be healthy in another century.

Evolutionary Medicine: Gloom and Hope

Evolutionary medicine is a sobering science. It reveals the many obstacles we face in fighting diseases, from the rapid evolution of antibiotic resistance to the deep vulnerabilities that make us susceptible to illnesses. But evolutionary medicine can also offer researchers inspiration and guidance in the struggle to improve our health. Models of evolution can reveal which antibiotic treatments will lead to more resistance, and which treatments will lower it. Scientists are also building similar models to help avoid the evolution of tumors that are resistant to chemotherapy and the evolution of HIV strains that resist antiviral drugs.

Evolutionary biology can also point scientists to important clues to new kinds of treatments. Ed Marcotte's experience with blood vessel genes in yeast is a case in point (McGary et al. 2010). Although scientists have sequenced the entire human genome, they still don't know the functions of many of our genes. Determining those functions demands a lot of time and labor as scientists manipulate individual genes in mice and then observe the changes that arise.

Marcotte wondered if looking at the ancient history of our genes might speed up the rate of discovery. As we saw in Chapter 8, networks of genes can take on new functions in different lineages. The same genes that determine the head-to-tail axis of the body become active in our arms, determining the anatomy from the shoulder to the fingertips. Marcotte wondered if he might be able to find gene networks in other species that also worked together in our own bodies.

Marcotte and his colleagues first created a database of scientific studies linking human genes to diseases. They reasoned that genes associated with the same disease normally belonged to the same network carrying out some function in the body. All told, they amassed 1923 of these associations.

Next, they turned to studies on other species. Scientists have systematically mutated many genes in organisms such as yeast and nematode worms and then noted how those mutations change their phenotype. Again, Marcotte and his colleagues reasoned that when several mutated genes altered the same trait, it meant they probably belong to the same gene network. They cataloged over 100,000 of these associations between genes and traits this way.

Then Marcotte and his colleagues started to compare the networks in different species. They found that the same genes clustered together in different species and were involved in different tasks. For example, they found five genes involved in cell-wall repair in yeast that matched five genes in vertebrates that are involved in repairing blood vessels.

This overlap suggested that the two clusters descend from a common ancestor of vertebrates and yeast. The scientists noticed that the cell-wall gene network in yeast contains a total of 67 genes, including the ones that were also known to be involved in blood vessels in vertebrates. Perhaps, they thought, the other 62 yeast genes also had counterparts in the blood vessel network.

It turned out that the 62 corresponding genes in vertebrates were fairly mysterious. To find out what their functions are, the scientists began running experiments on frog embryos. They would inactivate each gene on the list and then observe how the development of the embryos changed. They began to find that the embryos grew defective blood vessels or experienced internal bleeding. As they had predicted, the genes belonged to the blood vessel network. So far, they've identified eight such genes.

This method of investigating genes turns out to be much faster than picking them out at random to test their functions. An evolutionary approach makes it possible to quickly take advantage of the vast amount of research on other species to better understand our own.

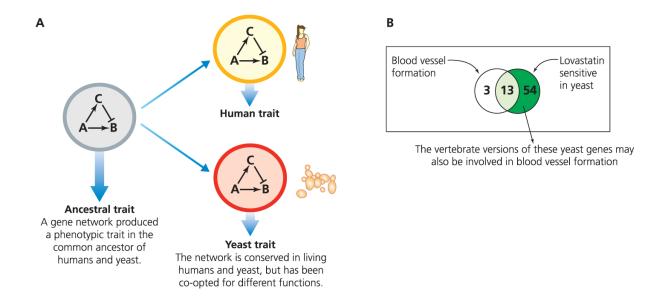
Marcotte's evolution-driven approach also speeds up his search for drugs to target the genes he's discovered. If searching for the functions of genes is like looking for a needle in a haystack, searching for a drug that cures a particular disease is like looking for a needle under a mountain of hay. There are trillions of trillions of compounds left for chemists to investigate.

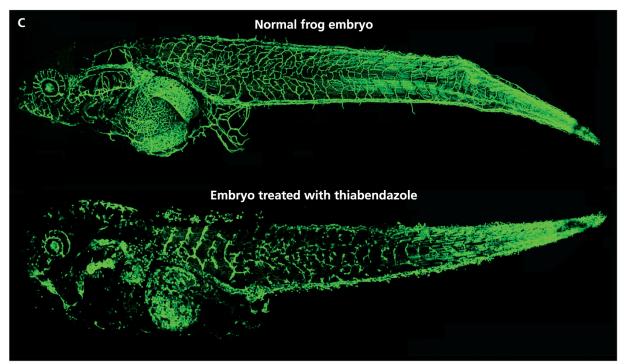
Scientists have tested a vast number of drugs on yeast. Marcotte suspected that a drug that attacks the yeast cell-wall cluster would also attack blood vessels in cancer cells. His graduate student Hye Ji Cha programmed a computer to search through millions of test results of different drugs on yeast.

She found a handful of molecules that targeted the cluster of genes that builds cell walls (Cha et al. 2012).

One of those molecules made the whole team of scientists excited. Known as thiabendazole, it was approved by the Food and Drug Administration for fighting fungal infections back in 1967 and was still on the market. That meant that the drug was safe. One of the biggest worries in the search for new drugs is that a promising compound will turn out to have toxic side effects. Thiabendazole's long track record made it unlikely that Marcotte and his colleagues would get such an unpleasant surprise.

Marcotte and his colleagues then tested their hypothesis on frog embryos. When Marcotte and his colleagues gave thiabendazole to the tadpoles, their blood vessels disintegrated into free-floating cells (FIGURE 15.19C). And as soon as the scientists washed the drug out of the tadpoles, they immediately started rebuilding their blood vessels. Marcotte and his colleagues then tried out thiabendazole on human blood vessel cells in culture. Normally, the cells organize themselves into tubes. But when exposed to the drug, they fell apart.





Zimmer, *The Tangled Bank*, 2e, © 2014 W. H. Freeman and Company Edward Marcotte.

FIGURE 15.19

Edward Marcotte and his colleagues took advantage of the deep homology of gene networks to discover new disease-related genes. A: Gene networks are surprisingly conserved over hundreds of millions of years. But in that time, they have been recruited to new functions. B: Marcotte and his colleagues searched for significant amounts of

overlap between networks of genes in vertebrates and networks of genes in other organisms. For example, they analyzed 67 genes that are associated with sensitivity to a drug called lovastatin in yeast. (Lovastatin interferes with the construction of the cell wall.) They discovered that in vertebrates, five of those genes were known to be essential for blood vessel formation in mice. Later experiments revealed that an additional eight genes also help build blood vessels. C: Marcotte and his colleagues discovered a drug called thiabendazole that interferes with a protein made by one of the yeast genes. They hypothesized that it would also interfere with blood vessel formation. The figure on the top shows a normal frog embryo, its blood vessels highlighted in green. At the bottom is a frog embryo treated with thiabendazole. This discovery suggests that the drug may be able to cut off blood from tumors and kill cancer cells. (Information from McGary et al. 2010 and Cha et al. 2012.)

These experiments on embryos gave Marcotte and his colleagues the confidence to try out thiabendazole on tumors. They coaxed human cells to develop into tumors, which they then transplanted into mice. Left untreated, the tumors grow rapidly, fueling their growth by coaxing the mice to build blood vessels. In mice treated with thiabendazole, however, the tumors grew much more slowly. After 27 days, the drug-treated tumors were only about a quarter of the size of the untreated ones. Marcotte is following up on this success with further experiments.

Medical researchers like Marcotte are like treasure hunters, wandering across a continent in search of jewels. A real treasure hunter wouldn't embark on the search without a map. Evolutionary biology offers medical researchers a map of life.

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Acheulean technology

Tools associated with hominins between 1.7 million years ago and 100,000 years ago. These tools are found across Africa, much of western Asia, and Europe. They are often found in association with *Homo erectus* remains. Acheulean tools, which include oval hand axes, display more sophistication in construction than Oldowan tools.

adaptive radiation

A rapid diversification of a lineage, accompanied by the evolution of species adapted to new ecological habits and environments.

allele

A version of a gene found in a population with a different sequence of bases than is found in other alleles.

allopatric speciation

The evolution of new species through geographical isolation.

allopatry

The geographic separation of two populations or species, which can provide the opportunity for reproductive isolation to evolve.

allopolyploidy

The evolution of new species by the duplication of genomes.

altruism

Altruism occurs whenever a helping individual behaves in a way that benefits another individual at a cost to its own fitness.

amino acid

A building block of proteins.

animals

Multicellular organisms that have to take in food to digest it. Compare with *fungi* and *plants*.

antagonistic coevolution

A form of coevolution in which one or both partners lowers the fitness of the other species.

antibiotic resistance

The ability of bacteria to survive exposure to an antibiotic.

antibiotics

Molecules produced by bacteria, fungi, and other organisms that can kill or inhibit the growth of bacteria.

Archaea

One of the three major lineages of life. Archaeans are single-celled microbes similar to bacteria.

asexual reproduction

Reproduction without sex.

Australopithecine

Members of the hominin genus Australopithecus.

background extinctions

Extinction rates that are not significantly higher or lower than the average rate of extinction.

Bacteria

One of the three major lineages of life. Bacteria are single-celled organisms that are common in soil, in water, and inside larger organisms.

balancing selection

A form of natural selection that favors more than one allele. It acts to maintain genetic diversity in a population by keeping alleles at frequencies higher than would be expected by chance or mutation alone.

base

Part of a nucleotide of DNA. There are four bases, which serve as an "alphabet" for genes.

bilaterian

An animal belonging to a lineage that includes vertebrates, insects, and worms. Bilaterian animals have many traits in common, including left–right symmetry.

biogeography

The study of the geographical distribution of species.

biological species concept

A concept of species defined by Ernst Mayr as "actually or potentially interbreeding populations which are reproductively isolated from other such groups."

biomarkers

Individual molecules from organisms that lived billions of years ago and that can be used to identify them.

BMP4

Bone morphogenetic protein 4, a protein that regulates the development of many structures in animals, including bird beaks and the dorsal—ventral axis of bilaterians.

body plan

A set of anatomical features found in all species in a lineage. The vertebrate body plan, for example, includes left–right symmetry, a skeleton, a skull, and a brain.

Burgess Shale

A fossil site in the Canadian Rockies that has yielded vast numbers of fossil animals dating back 505 million years.

chromosome

A threadlike package of DNA and associated proteins. Humans have 46 pairs of chromosomes.

clade

A group of organisms that share a common ancestor. In some cases, a clade corresponds to a traditional classification, such as a species, an order, or a class.

cladogram

A diagram of the distribution of shared derived characteristics among a group of related organisms.

cnidarian

An ancient lineage of animals that today includes jellyfish, anemones, and corals. Cnidarians lack brains and have a unique spear-like weapon called a nematocyst.

codon

Three consecutive bases that specify an amino acid in a protein.

coelacanth

A lobe-fin closely related to lungfishes and tetrapods.

coevolution

The evolution of two or more ecological partners as they adapt to each other.

coevolutionary arms race

An antagonistic form of coevolution in which species evolve stronger toxins, weapons, or defenses against each other.

continuous variation

Variation in a trait, such as height, that varies smoothly through a population.

convergence

The independent evolution of similar traits in separate evolutionary lineages.

convergent evolution

The independent origin of similar traits in separate evolutionary lineages.

cospeciation

The parallel speciation of coevolutionary partners.

derived trait

Describes a trait that is shared only by species within a clade. Hair is a derived trait of mammals. All mammals have eyes, but eyes are not a derived trait of mammals because other animals have eyes as well.

diapause

A kind of suspended animation.

diffuse coevolution

A form of coevolution in which each species has several coevolutionary partners—such as a flower that is pollinated by several species of insects.

dilution effect

The safety in numbers that arises through swamping the foraging capacity of local predators.

directional selection

A form of natural selection that favors individuals at one end of a range of a phenotypic trait. Directional selection can drive long-term change in a population (such as a gradual increase in body size).

discontinuous variation

A trait that is found in two or more discrete forms, such as the wrinkled or smooth surfaces of peas.

disruptive selection

A form of natural selection in which the mean value of a trait has the lowest fitness in a population. Disruptive selection favors individuals at

either end of a distribution of a trait. If the fitness of medium-sized individuals is low, for example, the frequency of large and small individuals will increase.

disparity

The morphological variation among a group of organisms.

dispersal

The spread of species to new ranges, such as when birds colonize distant islands.

DNA

Deoxyribonucleic acid, which stores genetic information in almost all organisms (with the exception of RNA viruses).

dominant trait

A trait whose production requires only one copy of the same allele. Compare to *recessive trait*.

Ediacaran fauna

Organisms that left some of the earliest known animal fossils. Some Ediacarans probably belong to the same lineages as living animals. Others may belong to lineages that have since become extinct.

Eukarya

One of the three major branches of life. Eukaryotes include animals, plants, fungi, and single-celled protozoans. All eukaryotes share certain traits, such as a nucleus.

eutherian

Mammals that bear live young and feed their embryos in the uterus. This branch includes humans and all other mammals that develop a placenta. See also *marsupial*, *monotreme*, *therian*.

evogram

A diagram that combines phylogeny with information from the fossil record to illustrate how a suite of traits (such as feathered flight) evolved.

evolution

Descent with modification.

exaptation

A trait that originates to perform one function and is later co-opted for another function.

extinction

The termination of a species or a lineage.

fitness

The success of an organism in its environment, which allows it to spread its genes in the next generation.

fixation

The process by which one particular allele spreads through a population over the course of generations, until no other allele of the same gene remains.

foramen magnum

The hole where the spinal cord exits the back of the skull. The downward orientation of this trait sets hominins apart from other apes.

fossil

A mineralized remnant of an organism preserved in rock.

fungi

Multicellular organisms that release enzymes to break down food before absorbing it. Compare with *animals* and *plants*.

gametes

Sex cells; egg cells and sperm cells.

genes

Segments of DNA that together encode a protein or an RNA molecule.

gene duplication

A mutation that produces an extra copy of a gene.

gene flow

The spread of genes across the geographical range of a population, or from one population to another.

gene recruitment

An evolutionary transition in which a gene begins to be expressed in another organ, in another pathway, or in some other context.

genetic bottleneck

The loss of genetic diversity through a rapid reduction in population.

genetic code

The set of rules governing the translation of any particular trio of bases into specific amino acids.

genetic drift

A change in the frequency of an allele owing to random sampling errors in a population.

genome

The full set of genetic information in a cell.

genotype

The genetic makeup of an organism; usually contrasted with the phenotype.

geographic mosaic theory of coevolution

A theory that proposes that the geographic structure of populations is central to the dynamics of coevolution. The direction and intensity of coevolution varies from population to population, and coevolved genes from these populations mix together as a result of gene flow.

germ-line mutation

A mutation that occurs during the development of gametes, which can be passed down to offspring.

green beard effect

A situation in which an allele (or a linked suite of alleles) produces three things—a recognizable phenotypic trait (the "green beard"), the ability to recognize the trait in other individuals, and preferential treatment of individuals with the trait.

group selection

Differential performance (fitness) of individuals causes some groups to outcompete and replace other groups.

Hardy-Weinberg principle

The frequencies of genotypes will be constant in a population from generation to generation unless they are disturbed by natural selection or some other influence, such as drift, immigration, emigration, and nonrandom mating. A population in this constant state is said to be in Hardy–Weinberg equilibrium.

heritability

The fraction of variation in a trait found in a population that is due to genetic factors.

hermaphrodites

Individuals that produce both female and male gametes.

heterozygote

An individual that carries two different alleles of the same gene.

hominid

Members of the clade containing humans (*Homo*), chimpanzees and bonobos (*Pan*), gorillas (*Gorilla*), and orangutans (*Pongo*). Commonly referred to as great apes.

hominin

Members of the "human" branch of the hominid clade, including the genus *Homo* and its close relatives such as *Australopithecus*, but not *Pan*, *Gorilla*, or *Pongo*.

Homo sapiens

The human species.

homology

Similarities in phenotype or genotype that are the result of common descent.

homozygote

An individual carrying two copies of the same allele of a gene.

horizontal gene transfer

The transfer of genetic material from one individual to another through a process other than heredity.

host races

Two sympatric—but genetically distinct—populations.

Hox genes

A set of genes that regulates the development of animals. They are important for defining the head-to-tail axis of bilaterians.

hybrid swarm

A group of highly interbreeding species.

hygiene hypothesis

A hypothesis that proposes that the normal development of the immune system requires the presence of a wide range of bacteria and other organisms in the body during childhood, and that the lack of early childhood exposure can raise the risk of autoimmune disorders later in life, such as allergies and asthma.

inarticulate

Having no hinge connecting the two shell valves.

inclusive fitness

A measure of the fitness of an individual that takes into account the extra reproductive success that relatives may have due to the phenotype of the individual. For example, individuals that help their siblings raise offspring may have a higher inclusive fitness than individuals that try and fail to raise offspring of their own.

individual selection

Differential performance (fitness) of individuals causes some genotypes to outcompete and replace other genotypes.

isotope

An atom, with specific reference to its number of neutrons.

lobe-fins

A lineage of vertebrates that includes tetrapods and their closest living aquatic relatives, including *Tiktaalik*, lungfishes, and coelacanths.

lungfish

A freshwater lobe-fin found in Africa, Australia, and South America. Lungfishes are the closest living relatives of tetrapods.

mammal

An animal such as a human, a kangaroo, or a platypus, with a set of shared traits, including hair or fur, and the ability of females to produce milk to nourish their young.

marsupial

Mammals that bear live young and develop a pouch to carry their young until they can survive on their own. This branch of mammals includes opossums, kangaroos, and the koala. See also *eutherian*, *monotreme*, *therian*.

mass extinctions

Widespread extinctions of species that occur in a large pulse lasting for a few million years or less.

meiosis

Cell division that gives rise to sperm cells or egg cells. During meiosis, the number of chromosomes per cell is cut in half.

melanosome

A cellular structure that absorbs light and helps produce color.

microRNA

A type of RNA that regulates gene expression after DNA is transcribed into messenger RNA. MicroRNAs bind to complementary sequences on specific messenger RNAs and can enhance or silence the translation of genes. The human genome encodes more than 1000 of these tiny RNAs.

mitochondria

Sausage-shaped structures that generate most of the energy in eukaryotic cells. Mitochondria probably evolved from free-living bacteria.

mobile elements

Segments of DNA that can be copied and pasted into other parts of the genome. Many mobile elements evolved from viruses.

monogamy

A mating system in which a male animal mates with only one female.

monotreme

Mammals that lay eggs and produce milk for their young. This branch includes the duck-billed platypus and the echidna. See also *eutherian*, *marsupial*, *therian*.

morphology

The form, shape, or structure of an organism.

mutualism

An ecological relationship in which two species depend on each other, such as sap-feeding insects and the bacteria inside them that synthesize amino acids that the insects can use.

natural selection

The process by which individuals better adapted for their way of life in their environment preferentially survive to leave more offspring with their traits to future generations.

Neanderthals

A population of *Homo sapiens* (or possibly a closely related species of hominids) that lived in Europe and Asia from about 200,000 to 28,000 years ago. Neanderthals were the closest relatives of living humans.

negative selection

The elimination of harmful alleles from a population through natural selection.

nonsilent substitution

A point mutation that changes the amino acid that is encoded by a codon.

notochord

A flexible, rod-shaped structure found in the embryos of all chordates. Notochords served as the first "backbones" in early chordates, and in extant vertebrates the embryonic notochord becomes part of the vertebral column.

nucleotide

A unit of DNA or RNA.

okenane

A molecule produced only by purple sulfur bacteria.

okenone

A red pigment that purple sulfur bacteria produce from okenane. See also biomarkers.

oncogene

A gene that transforms a cell into cancer under certain circumstances.

orthologs

Homologous genes separated by a speciation event (as opposed to paralogs, which are homologous genes produced by gene duplication that are both possessed by the same species).

p53

A protein that suppresses tumors by causing cells with abnormal cell cycles to die or to stop dividing.

parallelism

Convergent evolution that results from mutations to the same genes in different lineages.

parasitism

An ecological relationship in which a parasite lowers the fitness of a host in order to raise its own fitness.

phenotype

The manifestation of a genotype.

phylogenetic species concept

The concept of a species as the smallest clade of organisms sharing a set of distinctive traits.

phylogeny

The evolutionary history of a group of organisms.

plants

Multicellular organisms evolved from green algae that produce their own food by photosynthesis. Compare with *animals* and *fungi*.

plasmids

Molecule of DNA, found most often in bacteria, that can replicate independently of chromosomal DNA.

plastid

A structure found in plants, algae, and other organisms that carries out photosynthesis. Plastids evolved from free-living bacteria.

pleiotropy

The influence of a single gene on several phenotypic traits.

point mutation

A single base changes from one nucleotide to another (also known as a substitution).

polyandry

A mating system in which females mate with multiple males.

polygamy

A mating system in which males mate with multiple females.

polygyny

A mating system in which a single male has many female mates at a time.

population genetics

The study of alleles in populations and how they change over time.

positive selection

The process by which new advantageous genetic variants sweep a population.

postzygotic isolation

The isolation of two populations through mechanisms that act after eggs are fertilized. For example, hybrids between some species are sterile.

prezygotic isolation

The isolation of two populations through mechanisms that act before eggs are fertilized. For example, females of one species may not be attracted to the songs of males from another species.

promiscuous proteins

Proteins that can carry out more than one kind of chemical reaction.

protein

A molecule assembled from amino acids.

protists

Single-celled eukaryotic organisms.

pseudogene

A gene that has mutated so much that it can no longer encode a protein.

punctuated equilibria

A model of evolution in which stasis and random walks are most common, with rare bursts of significant change.

random walk

A series of random steps. The changes that occur in many lineages are random walks, as opposed to directional change or stasis.

recessive trait

A trait whose production requires two copies of the same allele. Compare to *dominant trait*.

recombination

The shuffling of chromosome segments during meiosis. Recombination is a major source of genetic variation in populations of sexually reproducing organisms.

reinforcement

The increase of reproductive isolation between populations through the selection against hybrid offspring.

relative fitness

The ratio between the average number of offspring produced from one genotype and the average number produced from another genotype.

retrovirus

A virus that encodes its genes in RNA and can insert its genetic material into a host's DNA. In some cases, retroviruses become integrated into the genomes of their hosts for millions of years.

ribosome

A set of RNA molecules and proteins that assemble amino acids into proteins.

ring species

A connected set of populations of a species with a range that brings the two end populations into contact without interbreeding.

RNA (ribonucleic acid)

A single strand of nucleotides. RNA molecules serve as templates of genes for making proteins. They also play other roles, such as shutting down the expression of other genes.

Sahelanthropus

The oldest known fossil of a hominid, estimated to have lived between 6 and 7 million years ago.

sauropods

A clade of dinosaurs characterized by extremely long necks and which produced the largest land animals known.

sexual conflict

A situation in which the optimal reproductive strategy for males comes into conflict with the optimal strategy for females. Sexual conflict can lead to males and females evolving strategies that lower the fitness of their mates.

sex ratio

The ratio of males to females in a population.

sexual reproduction

A mode of reproduction in which gametes are combined. Though bacteria are asexual, some researchers consider horizontal gene transfer a form of sexual reproduction.

sexual selection

Changes in the gene frequencies of a population caused by different levels of mating success of different genotypes.

silent substitution

A point mutation that does not change the amino acid encoded by a codon.

somatic mutation

A mutation that occurs in a cell in the body of an organism, as opposed to a mutation in a gamete.

speciation

The evolution of new species through splitting from an ancestral lineage.

species

A lineage made of linked populations.

stabilizing selection

A form of natural selection in which the average individuals in a population have higher fitness than ones at either end of a trait's range. Stabilizing selection can keep a trait from changing in a population.

stasis

A period of thousands or millions of years in which a lineage undergoes little or no directional change in a particular trait.

stromatolites

Rock-like structures formed by layered mats of bacteria. While stromatolites are rare today, they were a common feature of the early Earth.

sympatric speciation

The divergence of individuals in a single area into two species.

sympatry

Occurs when populations are in the same geographic area.

synapsids

A clade of tetrapods that emerged 300 million years ago and that includes the mammals.

taxonomy

The science of classifying organisms by the use of reliable characteristics.

teleosts

A large clade of bony fishes that includes the most familiar species, such as tuna, salmon, and goldfish.

tetrapods

A clade of vertebrates that includes mammals, birds, reptiles, and amphibians. Tetrapods evolved from lobe-fins about 360 million years ago, acquiring limbs and other traits that allowed them to live on land.

theory

An overarching set of mechanisms or principles that explain a major aspect of the natural world.

therian

Mammals that bear live young rather than laying eggs; therians form two of the three living branches of mammals. See also *eutherian*, *marsupial*, *monotreme*.

trackway

A series of fossil footprints.

transcript

The sequence of a messenger RNA molecule transcribed from a gene.

transcription

The process that takes place when RNA polymerase reads a coding sequence of DNA and produces a complementary strand of RNA, called messenger RNA (mRNA).

translation

The process that takes place when a strand of mRNA is decoded by a ribosome to produce a strand of amino acids.

urbilaterian

The common ancestor of all living bilaterians.

vicariance

The splitting of a species or a group of species within a common ancestral geographic range. Vicariance can occur through the splitting of continents, the formation of new mountains, and other geological events.

virulence

The relative ability of a pathogen to cause disease.

virus

An infective agent consisting of DNA or RNA enclosed in a protein coat. Viruses can reproduce only by invading a host cell.

zircons

Microscopic crystals in which traces of the first few hundred million years of Earth's crust are preserved.

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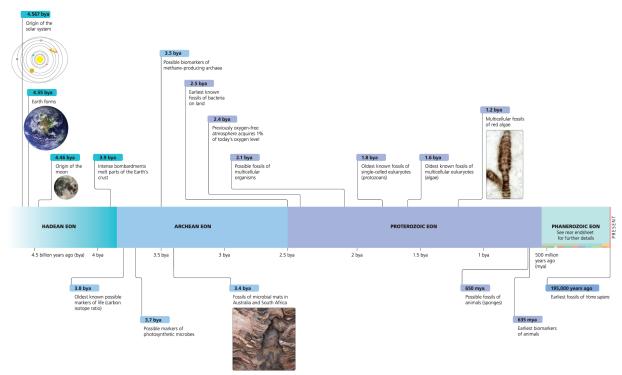
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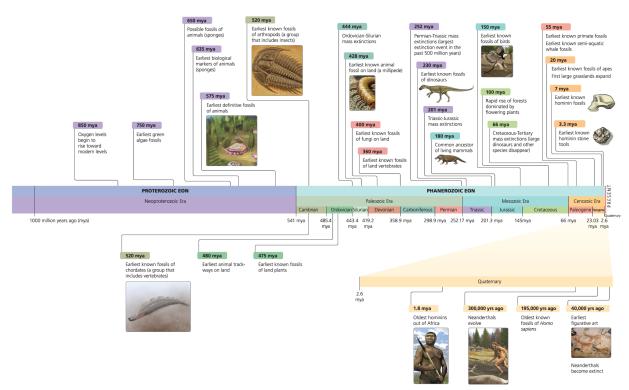
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